The American Journal of Surgery

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EDITORIAL

THE SOUTHERN SURGICAL ASSOCIATION: AN APPRECIATION

THE Transactions of the forty-seventh annual meeting of the Southern Surgical Association held last December (1934) at Sea Island, Georgia, which appears in this journal, will exhibit the scientific activities of an organization which, since its foundation in 1887, has stood as the leading and most representative exponent of surgical thought and authority in the South.

When this volume is bound and placed side by side of its forty-six predecessors, it will add another unit to the cumulative library of surgical progress that the Association has contributed annually to the surgical literature of America, during the forty-seven years of its existence. A glance at the contents of this well-nourished volume will suffice to prove that the Association has not only lived up to the ideals and highest expectations of its founders, but has surpassed their fondest anticipations by expanding the sphere of its cultural influence far beyond its regional domain, and by becoming, as it is now, one of the most productive contributors to the progress and diffusion of Surgery in America, and a factor of no mean importance in advancing the scientific interests of the surgical guild throughout the world.

To the Writer, who has enjoyed the privilege of participating in the affairs and functions of the Association as one of its most attached veterans, the appearance of this splendid volume suggests many gratifying reflections. These tend to be retrospective and con-

verge toward the origin of the Association, the circumstances that attended its birth and the factors that gave it the initial impulse, the vitality and growing vigor which it has displayed ever since it began its remarkably harmonious and productive career. To attempt a discussion of the underlying causes that have given this Society its commanding position as the oldest and most authorized exponent of Surgery in the South, or to account for the notable characteristics that have given it its distinctive individuality and attractiveness, would involve a narrative which would soon drift into personal recollections and in a history which, no matter how aggreable to the historian, would be too lengthy to be compatible with the purpose of this introduction. Suffice to say in the words of one of its founders, that "while the Southern Surgical Association may be limited *geographically* by Mason and Dixon's line, *surgically* it embraces the entire Nation." To this may be added another sentiment which is true of the born Southern Surgeon who realizes, while enjoying the delightful conviviality ties of these meetings, that "though *Surgery* is *Surgery*, *Dixie* is *Dixie*, and there is no other Dixie to those who have been crooned to sleep by the soothing melody of her lullabies."

To all concerned in the production of this admirable record of the Association's activities, the undersigned, in grateful appreciation of the profit and pleasure that he has derived from them, extends his heartiest congratulations.

RUDOLPH MATAS.

TECHNIQUE OF OPERATION ON THE COMMON BILE DUCT

WITH SPECIAL REFERENCE TO INSTRUMENTAL DILATATION OF THE PAPILLA OF VATER*

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BOSTON, MASS.

THERE has been a definite trend in recent years to combine choledochostomy with primary operations upon the gall bladder. This is a natural tendency inasmuch as we are becoming more familiar with the signs, symptoms and operative findings leading us to suspect that the pathology is not confined to the gall bladder alone. The factors indicating common-duct exploration have been stressed by various authorities so that now there is a much higher ratio of choledochostomy at the primary operation than ever before. This has led to greater experience in this procedure with the development of the technique involved, so that a more satisfying operation is done from the standpoint of the surgeon and with a definite reduction in unrelieved patients and secondary operations. With better preoperative preparation, improvement in anesthesia, more careful surgical technique and rational after care, the mortality has not increased as much as would be expected by the added exploration of the duct. This is particularly evident when the type of case usually requiring choledochostomy is taken into consideration. It is yet a bit soon to have much to say regarding the ratio of late secondary operations following choledochostomy, but it is logical to deduce that when stones are removed from the common duct and the papilla of Vater properly dilated that there will certainly be a decided reduction in choledochostomy as a

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secondary procedure. Judd and Marshall[1] in a large series of common-duct explorations reported that stones were found in 1768 patients, representing 13.2 per cent of all patients operated upon for biliary tract stones during the same period. Seventy-five or 4 per cent of these had returned to the Mayo Clinic with recurring symptoms and stones were again removed from the common duct in 55 cases. Lahey[2] reports that the percentage of primary common-duct exploration has gradually increased in his clinic from 15.5 per cent in 1926 to 42.5 per cent in 1931, with a corresponding increase in stones found from 8.4 per cent in 1926 to 21.0 per cent in 1931. Approximately 50 per cent of all ducts explored yielded stones.

Although there have appeared in the literature a considerable number of references to the dilatation of the papilla of Vater at the time of choledochostomy, these have often referred to unusual circumstances, carried out with half lengths and various types of urethral sounds. At one time slight popularity of the method was gained for the purpose of instilling fluids into the duodenum, by a tube leading into it from the common duct (McArthur,[3] Matas[4]). Some articles, mainly foreign, quoted by Cheever[5] have dealt with dilatation of the papilla for the purpose of so-called perfect drainage of bile into its normal habitat rather than to the outside of the body through a tube leading from the duct. W. J. Mayo[6] advocated gentle instrumentation of the papilla in common-duct stone operations to allow for the passage of overlooked calculi. According to him, approximately one-third of all patients coming to autopsy following choledochostomy for stone would show stones in the ducts that had not been found at operation. Cheever realized the importance of dilating the outlet of the duct and experimented upon cadavers, finding that the normal papilla would admit an instrument the size of a No. 10 F. sound. He further demonstrated that careful dilatation could be carried out to Nos. 18 or 20 F. without apparent tearing of tissue. He practiced routine dilatation of the papilla when choledochostomy was done, utilizing for the purpose woven

silk urethral catheters. This made it possible for him to combine dilatation with irrigation of the duct. At the time of his publication, he had carried out this procedure 50 times in

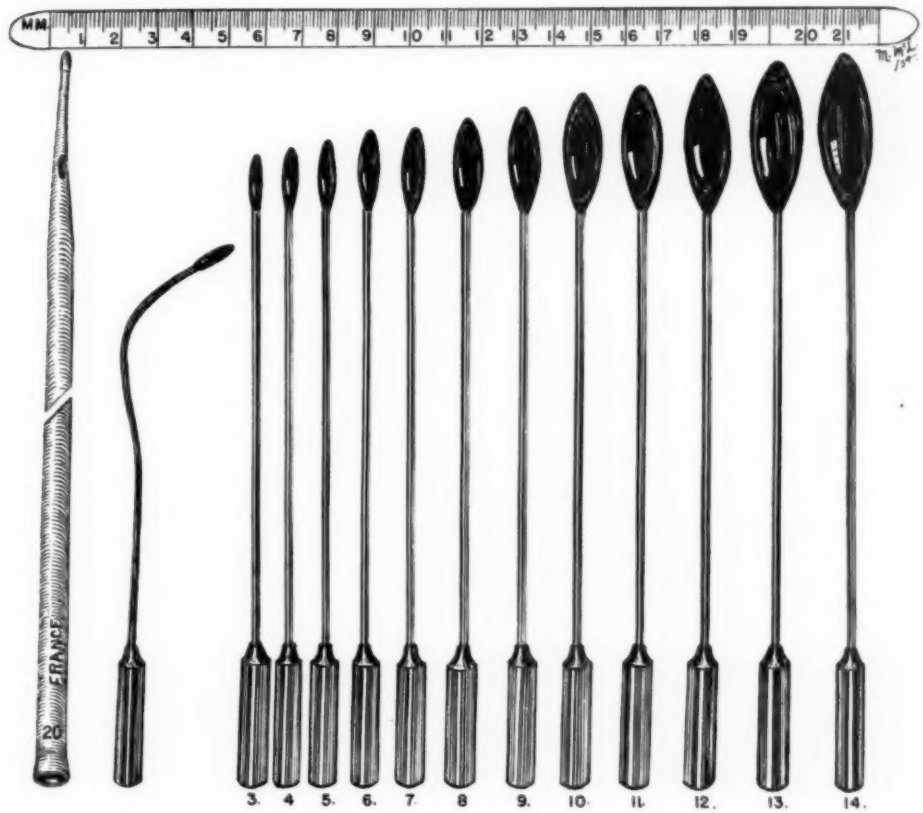

FIG. 1. Dilators with bendable handles as advocated by Bakeš. To left is shown a No. 20 silk woven catheter, the type used by Cheever.

300 operations upon the biliary tract without evidence of harm to the patient. Having seen Dr. Cheever's technique several years ago, we used it with considerable satisfaction until our attention was called to a method used and described by J. Bakeš[7] of Brünn, Czecho-Slovakia, and brought to us by Dr. Richard Chute of Boston, in 1930.

Bakeš believed that perfect drainage of bile could be established into the duodenum by a routine dilatation of the papilla when choledochostomy was practiced and also that this

dilatation permitted the passage of stones that were inaccessible within the hepatic ducts. He attempted to close the incision in the duct as tightly as possible but admitted that there

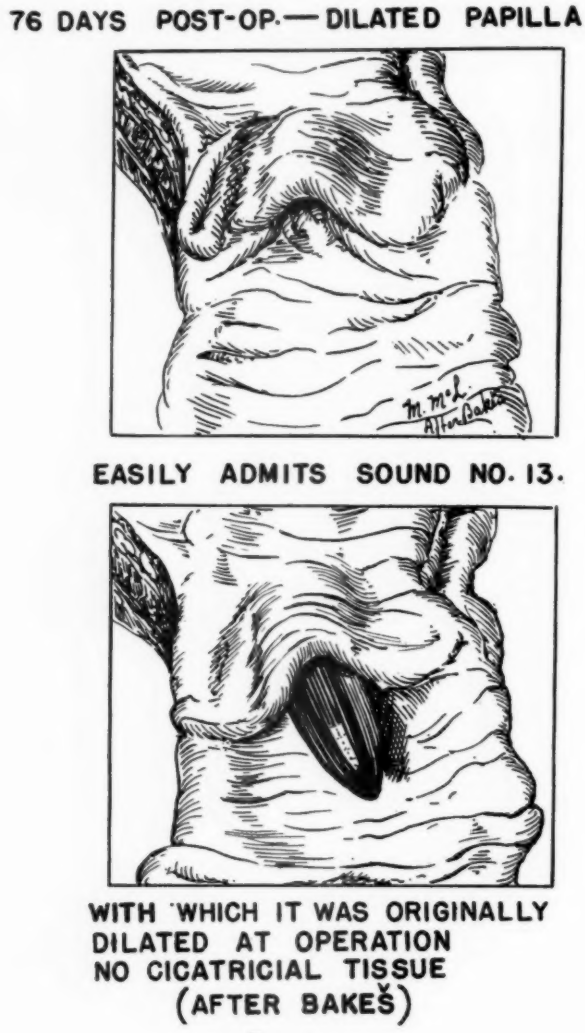

FIG. 2.

was considerable bile leakage along the drains for several days in most instances. He devised some rather long olive-shaped bougies with bendable handles for the purpose of gradual dilatation of the papilla (Fig. 1). These measure in diameter approximately from 3 to 14 mm. and are so numbered. Ordi-

narily he practiced dilatation of the papilla to approximately the diameter of the duct. He was inclined to believe that in no instance had any harm come to the patient by this method in

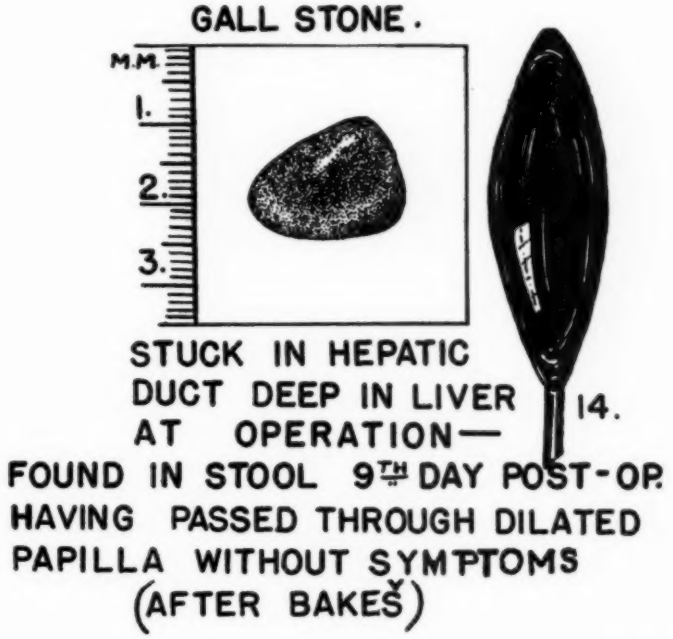

FIG. 3.

250 cases reported, although 22 of these patients had died following the operation. He further states that he believes the dilatation will last a long while and may be permanent. One patient died of hemorrhage from carcinoma of the stomach seventy-six days after operation. The papilla at autopsy (Fig. 2) admitted the 13 mm. sound with ease, the size to which the dilatation had been carried at operation. Sections made of the tissue after dilatation were said to contain little evidence of scar tissue. He illustrates one stone measuring 14 mm. in diameter that eluded him after he had dislodged it from the ampulla. He was not able to bring it out of the dilated hepatic duct. Dilatation was carried out to 14 mm., approximately the size of the duct. The stone was passed nine days afterwards without pain (Fig. 3).

We have not adopted the technique as described by either Cheever or Bakeš entirely but believe that some of the points described by each as well as certain details advocated by others are useful, and so have finally developed a more or less routine management of these cases.

TECHNIQUE OF OPERATION

The abdomen is opened through a long right paramedian incision. The rectus fascia is incised 1.5 cm. from the midline and the muscle retracted outward. The peritoneum is opened in a line parallel to the incision in the fascia. Adhesions are freed and in the absence of acute inflammation, the abdominal and pelvic viscera explored. Careful palpation of the pancreas is carried out and, if thickened, the posterior surface of the transverse mesocolon is inspected for fat necrosis. If malignancy in the head of the pancreas is suspected, the gall bladder is not removed until after the duct is explored. Usually, however, the gall bladder is decompressed with a suction trocar if distended. The ducts are then exposed as well as possible and the gall bladder removed from below upward if the exposure is satisfactory, but often from above downward. The cystic duct and artery are secured with separate clamps and great care is taken to leave flaps of the serous coat of the gall bladder to allow peritonealization of the gall-bladder bed. The duodenum is then freed if necessary so that the common duct is further exposed. The common duct is then aspirated with a small hypodermic needle and opened longitudinally in its supraduodenal portion, the escape of bile being taken up with a suction tip. Silk guy sutures are passed through each edge of the incision which is then carried distally so that its length is approximately that of the diameter of the duct. (Fig. 4.) The operator then goes to the left of the patient and by placing the fingers of the left hand beneath the duct and the thumb on the anterior surface, a careful palpation can be carried out under vision. Frequently stones can be gently manipulated along the duct and seized when they reach the

opening in it. The fenestrated forceps are then used in both directions with care. A probe is then passed through the papilla under direct palpation and the Bakeš dilators passed

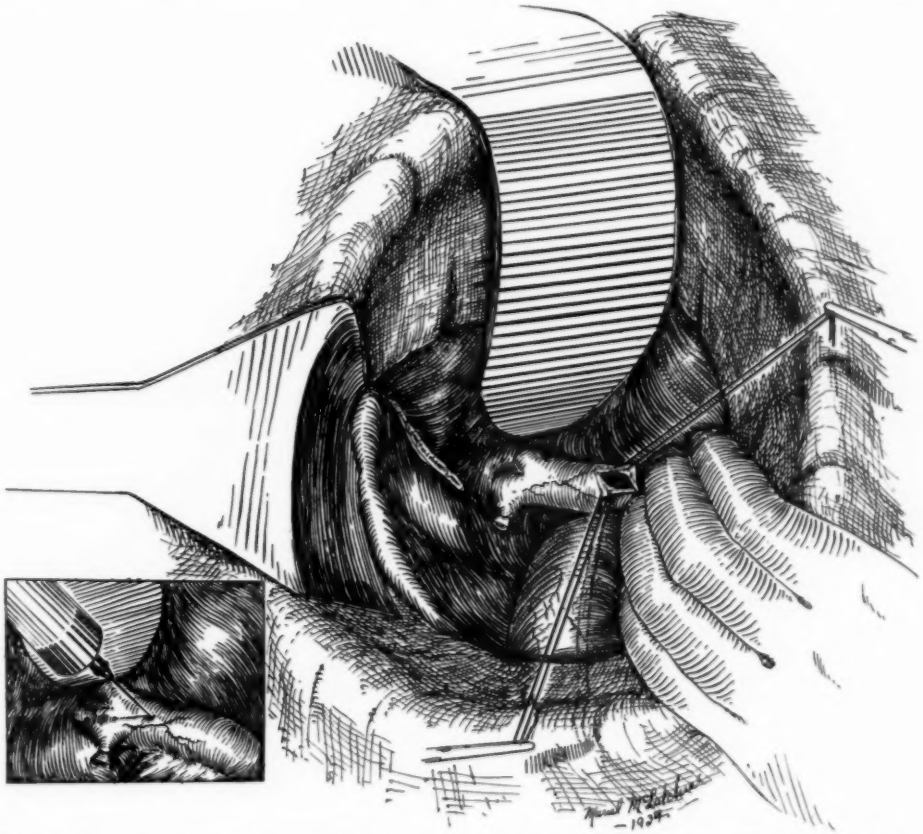

Fig. 4. Development of common duct with incision held open by silk guy stitches.

through in succession until the desired amount of dilatation is obtained. (Fig. 5.) As these dilators pass through the papilla they will give a slight jump easily discernible to the palpating hand. Very little force must be used. If it is impossible to safely pass the small probe through the papilla, the duodenum should be opened longitudinally over it and a retrograde dilatation started. After completion of the dilatation, the duodenum should be closed transversely. The dilatation may be carried out to the size of the duct in most instances with safety. Often the operator is satisfied to dilate the papilla to a

diameter sufficient to allow easy passage for stones of the size found in the gall bladder. When the dilatation is done with the duodenum open it is surprising to see how little apparent

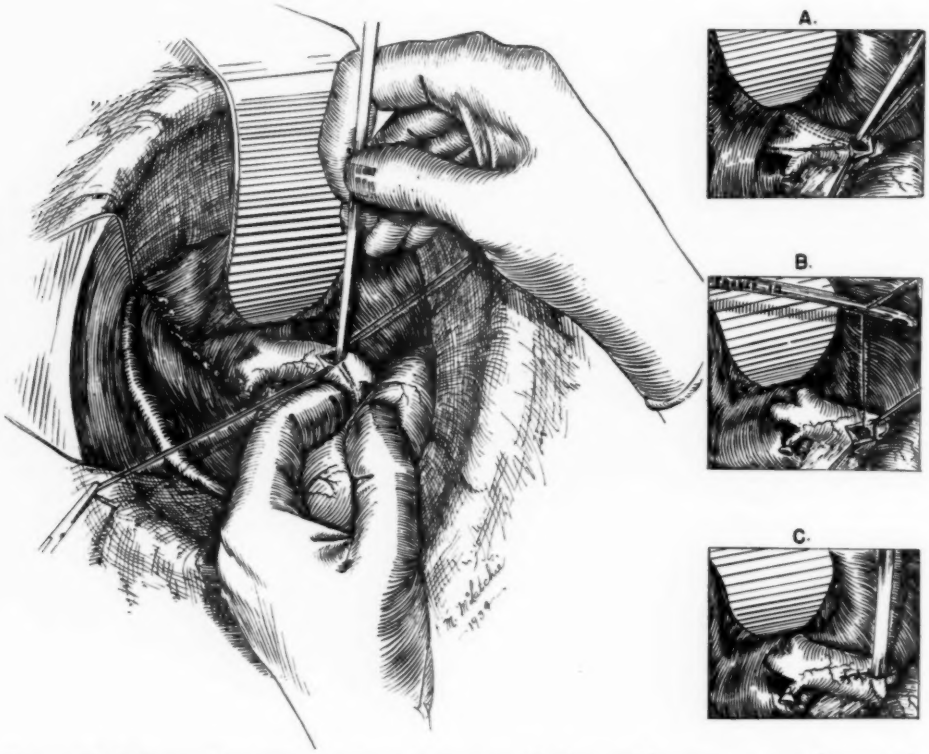

FIG. 5. Technique of instrumentation of common bile duct with operator standing to left of patient. This allows direct vision while these maneuvers are being carried out. Insets B and C: method of fixing catheter in duct.

injury is done to the tissues in this region. A gentle technique with rather slow dilatation, however, is recommended. Considerable care must be exerted not to tear the duct at the incision with an incorrectly bent handle or with too much force. If the ducts have many stones or sand, they are irrigated with normal salt solution. The gravel and debris can be picked up by a suction tip as advocated by Lahey.[8] Occasionally, stones have been removed from the region of the ampulla with a scoop or forceps after the dilatation has been accomplished. A No. 10 F. soft rubber catheter is sutured into the

lower angle of the opening in the duct with oo chromic gut on an atraumatic needle. This suture is utilized to close the rent in the duct, which is reinforced by the areolar tissue

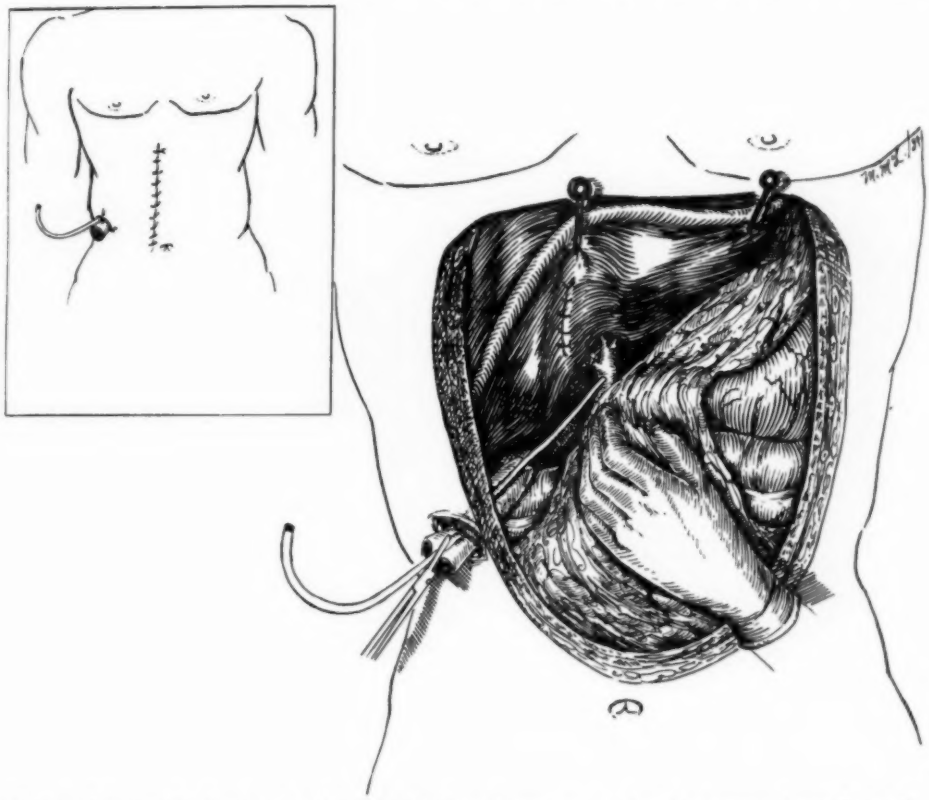

FIG. 6. Schematic drawing with abdominal wall cut away showing approximately position of short cigarette wick in subhepatic fossa (Morison's space) and catheter running into common duct, being separated from duodenum by omentum.

pushed aside in the original exposure of the duct. The cystic duct may be utilized for the catheter if it is of suitable size. Usually it is examined for debris and tied off with No. 1 chromic gut. The bed of the liver is peritonealized. The edges of the peritoneum, fascia and skin are held upward and toward the midline. A stab wound is placed at the lower border of the twelfth rib and through this is led, under direct vision, the catheter and a short cigarette wick, the latter resting in the subhepatic fossa (Morison's pouch) (Fig. 6). Care is taken to place the wick as far from the duodenum, ducts and gall

bladder bed as possible. Omentum is laid over the duodenum and the abdominal wound tightly closed. Bile drainage will average about 300 c.c. per diem through the catheter. There may be a little from the cigarette wick for a day or two. The wick is removed on the tenth postoperative day and the stitch that holds the catheter to the skin is cut the same day. Often the catheter will come away with the wick. If not, it will let go within forty-eight hours. After its removal there is very little bile leakage along the sinus which is dry within two or three days.

The smoothness of convalescence as evidenced by the lack of duodenal irritation, vomiting, etc. as well as the failure of residual pockets of bile and infected hematomata to collect, make us feel that drainage in this manner has much to commend it. The vertical incision with its better exposure of the duct region is thus combined with dependent drainage, one of the chief advantages credited to the transverse approach. It is our belief that the foregoing technique is attended with a lower incidence of infected wounds and incisional hernia, and may play a rôle in diminishing the severity of postoperative adhesions around the duodenum. Since this has been found satisfactory in a comparatively large group of patients, we feel justified in presenting it at this time. We do not wish to claim priority in any detail as we realize that many of the steps are in common use and that all of them have doubtless been employed. We wish, however, to stress the importance of routine dilatation of the papilla of Vater when the common duct is explored and urge a more careful consideration of the question of choledochostomy in a larger proportion of primary operations on the biliary system. Perhaps the simplicity of our technique and the results obtained may encourage a more common practice of the methods described.

INDICATIONS FOR EXPLORATION OF THE BILIARY DUCTS

At a risk of calling attention to well-known facts, it seems worth while to briefly outline the more common signs, symp-

toms and operative findings that make duct exploration not only justifiable but important. Many of these have been set down before, and more recently by Lahey[2,8] in an impressive manner.

(1) *Previous Cholecystectomy or Choledochostomy with Continuation or Recurrence of Symptoms.* Nothing is more disappointing to the surgeon than to have his patient fail to be relieved of the symptoms for which an operation is undertaken. Many such patients do not return to the surgeon doing the original operation but enough of them do to remind us that we must be charitable to our fellow surgeons who have failed at a previous procedure in many of the patients who come to us for a second, third or fourth attempt for relief. In such individuals, we must not be satisfied with lysis of adhesions or attempting to explain typical biliary pain on some other organ, such as the duodenum or pancreas. Often there will have resulted impairment of liver function or jaundice. In these cases we will usually find the common duct enlarged and may be able to palpate one or more stones within it. However, even if colesterol crystals are obtained by duodenal lavage in larger quantities than normal, we may not find a stone. The ampulla of Vater may be found constricted even to such a degree that a small probe cannot safely be introduced into the duodenum through the duct. In such instances, the duodenum should be opened and a retrograde dilatation of the papilla carried out. In one of the early attempts to employ mechanical dilatation in a patient, who had had previous cholecystectomy, a junior surgeon created a false passage into the duodenum. The catheter drained duodenal contents while bile leaked around the cigarette wick. After the wick and catheter was removed on the tenth postoperative day, suction was employed to prevent digestion of the wound. The external fistula healed promptly and the patient was discharged in good condition, about four weeks after operation. Although temporarily relieved of symptoms that were apparently due to constriction of the distal portion

of the duct rather than stone, we feel that a recurrence of symptoms will be more likely to take place than if the dilatation had been carried out through the papilla.

(2) *Jaundice at Time of Operation or in the Past.* The only deterrant from exploratory operation on such a patient should be a definite history and laboratory findings conclusively indicating a so-called medical jaundice. The borderline cases should be subjected to surgery as well as those in whom the history and findings indicate obstructive jaundice. If the jaundice is decreasing and the symptoms clearing during the time of investigation, we believe it safer to delay operation until the patient has ceased to improve. If the jaundice is stationary or increasing, then with proper preparation, operation should be undertaken with as little delay as is consistent with safety. One must not assume that inflammation extending from the gall bladder has produced a temporary constriction about the outlet of the duct but should explore and drain the duct. In this type of case we believe dilatation of the papilla of utmost importance, as without it there may result a leakage of most or all of the bile to a point of depletion and actual death. The removal of a stone impacted in the distal end of the duct is imperative and this alone is apt to be met with gratifying results. One must not overlook the fact, however, that other stones may lie within the dilated liver ducts and for this reason dilate the outlet sufficiently to allow the passage of such stones.

In one such instance, that of a man of fifty-nine operated upon after six weeks' illness beginning with colic followed by jaundice that had nearly cleared up, the papilla was dilated to approximately the size of the duct (9 mm.). No stone could be found within the duct. There was a good deal of thickening in the head of the pancreas. The catheter yielded an abnormal amount of bile reaching, on the fourth day, 2560 c.c. The stools on the fifth, sixth and seventh day were clay colored. The patient's condition became alarming, his appetite poor, his fluids hard to maintain. On the eleventh postoperative day after removal of the tube, he had a very severe attack of pain similar to his original episode. Following this the external drainage ceased and his convalescence from then on was uneventful. Unfortunately

through a misunderstanding with the nurse, the stone was not sought in the stool. We have little doubt that such a stone was overlooked at operation and that it completely obstructed the dilated outlet temporarily.

One may find the outlet constricted by scar tissue or by pressure from chronic pancreatitis and such cases should be dilated. It is important, however, not to confuse carcinoma of the head of the pancreas with inflammation as here we may lose an opportunity for cholecystgastrostomy and a long respite from jaundice.

(3) *History of Chills and Fever following Biliary Colic.* Here one must assume that there has been interference with the drainage of bile from the liver. The infection is often associated with stones in the ducts or an inflammatory constriction of the outlet into the duodenum. In either instance, the temporary drainage of the common duct will greatly enhance recovery of the liver. Dilatation of the papilla of Vater allows this infected bile to drain mainly into the intestine and shortens the time of bile loss to the patient. In some instances the duct will be contracted, especially in delayed operations; these will be taken up in more detail under Cholangitis. That drainage of the duct should be associated with cholecystectomy in these cases has been fairly well established, as the gall bladder is, as a matter of fact, usually the focus of infection. Drainage alone may be all that can be done for a very ill patient but a secondary operation in such instances must be anticipated. The two-stage operation is a justifiable procedure under proper circumstances and often carried out successfully when a complete operation in one stage may result fatally.

(4) *History of Very Frequent Attacks of Biliary Colic.* The frequency of attacks associated with common duct stones was first called to our attention by Dr. D. F. Jones,[9] who taught us much, concerning the details of the patient's symptoms in relationship to the pathology present. The patient who has attacks of pain once a week or more often is apt to have small stones in the gall bladder and when this is

true an exploration of the ducts will often yield stones. If, under these circumstances, a stone is not found within the duct, one cannot safely assume that such a stone has passed into the duodenum. Even if one or more stones are removed from the ducts, there may be others lodged in the hepatic ducts, that are inaccessible by the usual methods at our disposal. If we combine our search with dilatation of the papilla, we may reasonably hope for the passage of such stones during the convalescence. Even if the stones in the gall bladder are large or absent, we feel that the ducts should be explored if there has been a history of frequent attacks of colic. A single stone may lie within the duct acting as a ball valve and thus producing attacks of such frequency as to prevent the patient from eating with a resulting rapid loss of weight.

(5) *Small Stones or Sand Found in the Gall Bladder.* One may find perfectly uniform small stones filling the gall bladder. These stones may be of a definite shape and size, often numbering into the thousands. If these are less than 5 mm. in diameter, one may assume that with each attack, one or more of such stones may have passed through the cystic duct into the common duct. Whether they pass on into the duodenum is dependent upon the size of the outlet, or whether this structure has been slowly dilated by the passage of stones to accomodate itself to the emergency. Stones are found within the ducts with such frequency under these circumstances that we must assume their presence there and not only attempt to remove them but dilate the sphincter sufficiently to allow others to pass if they should be overlooked. In analysing the operative notes and the history of patients operated upon secondarily for common-duct stones, we find many instances of small stones found at operation and a history of frequent attacks of pain prior to the first operation. It is not safe to leave sand and debris within the stump of the cystic duct or within the common duct itself. Many secondary operations have been necessary in such cases. There may even be temporary relief of sufficient duration for one to credit the stones found in the

ducts at a second operation to a new development within the duct. As a matter of fact, these are probably more often the result of overlooked nuclei at the time of the first procedure.

(6) *Palpable Stones within the Ducts.* It would seem unnecessary to say more under this heading but the manner in which palpation is carried out is of utmost importance. Exposure of the common duct is not always easy, and frequently one may feel justified in not freeing the duodenum sufficiently to allow a careful palpation of this region. We believe, however, that in the majority of cases, sufficient exposure can be obtained, safely, to permit the operator to place the fingers of the left hand posterior to the region of the ducts and with the thumb on the anterior surface, a very satisfactory palpation can be carried out. This may be done with much greater ease and security if the surgeon will stand on the left side of the patient. This maneuver is of greatest help in the manipulations necessary after the duct has been opened. One must not be content with a casual thumb and finger palpation not under direct vision as a stone may slip along the duct in an elusive manner. Also, thickening in the head of the pancreas may often make it impossible to be sure of the transduodenal portion of the duct. It is our belief that the duct should be carefully palpated routinely and explored if there is even a slight suspicion of trouble within it.

(7) *Dilated Common Duct.* It is usual to find the common duct increased in its diameter at secondary operations, particularly after cholecystectomy. If looked for, it will be found in a very considerable number of cases at the primary operation. This is particularly true in biliary tract disease of long standing. Without doubt, the gall bladder in many cases is completely shut off from its normal connection with the ducts, due to long impaction of cystic-duct stone with secondary inflammation. This may be present in any degree or such a condition may have existed in the past sufficiently long to reproduce the enlargement found in the duct after cholecystectomy. There may or may not be other signs and symptoms

indicating the possibility of the presence of common-duct stones. We believe that one should explore every dilated duct regardless of the sequence of events or the evidence at hand in the individual case. Stones are often found in such ducts and when not present the outlet of the duct is nearly always found to be constricted and more difficult to probe than the average. Dilatation of the papilla should usually be carried out under these circumstances to a diameter larger than the stones removed. Some duct stones are so large, however, that we have not dared or thought it necessary to carry the dilatation to the diameter of the duct as suggested and practiced by Bakeš. In a few instances we have carried the dilatation up to No. 13 Bakeš without harm to the patient. In certain cases when the duct was enormous, it has not been drained in the usual manner, but in spite of dilatation of the outlet and a careful suture of the duct, there has always been leakage of bile around the cigarette wick. This makes us feel that even in large ducts, the routine practice of a small catheter leading from the duct to the outside has its advantages.

(8) *Thickening in the Head of the Pancreas.* One so frequently finds abnormal thickening in the head of the pancreas and occasionally throughout its length associated with biliary tract disease, that often the question arises regarding the type and degree of pancreatitis present. In such cases, it is wise to acquaint one's self with the condition of the intra-abdominal fat and particularly the posterior portion of the transverse mesocolon. If no fat necrosis is found, one may assume that the pancreatic thickening is not an indication of the hemorrhagic type of inflammation. One may term this thickening in the pancreas, chronic pancreatitis, but may regard it in a less serious light than the term pancreatitis usually implies. At any rate, it is usually secondary to the inflammation and obstruction in the biliary tract. This complication makes the accurate palpation of the transduodenal portion of the common duct difficult, and is apt to be associated with considerable inflammation within the duct

itself. The walls of the duct are apt to be thickened and its lumen may vary from a markedly decreased diameter to considerable dilatation, which depends on the duration of the disease and Nature's efforts to cope with it. When the duct is large, a satisfactory dilatation of the outlet can be carried out without, in the majority of cases, flaring the chronic inflammation into a more fulminating type of pancreatitis. The one patient, reported in our deaths, who developed pancreatitis following dilatation had no evidence of any pathology in the pancreas at the time of exploration. We have so often found a stone impacted in the ampulla in these cases with thickening in the pancreas, that we feel certain that the chance of a successful outcome far outweighs any argument against manipulation under these circumstances. On a few occasions we have been able to remove a fragmented stone from the ampulla after dilatation has been completed.

(9) *Contracted Gall Bladder.* When biliary disease is of such long standing or of such a character as to completely and permanently occlude the cystic duct, the contracted gall bladder results. Doubtless many patients who reach this condition will have permanent relief from biliary colic. Also, in time, the ordinary digestive upsets secondary to gall-bladder disease may cease. It therefore follows, that if such a condition is found at operation in a patient whose symptoms are those of biliary tract disease, that one may expect to find the disturbing element in the biliary passages. This condition is often found in obstructive jaundice and varying degrees of cholangitis are apt to be present. It is in this type of patient that we expect to find stones in the ducts and should they not be located we feel that simple exploration and drainage is apt to be met with failure. We believe that dilatation of the papilla offers better drainage of the duct into the duodenum and may allow the passage of an elusive stone held within the hepatic ducts.

(10) *Gall Bladder without Stones.* In a recent article by Graham and Mackey,[10] the so-called minimal diseased

gall bladder yielded a very high percentage of unrelieved patients after cholecystectomy. In an individual with typical biliary symptoms with or without a positive Graham test, one may find a gall bladder of almost normal appearance without stones. Even if real cholesterosis is found the true cause of the symptoms may be found within the ducts. These ducts should be explored whether they are of normal size or dilated. In them we are apt to find debris or well-formed stones or the outlet may be constricted to a degree indicating that drainage of bile into the bowel has definitely been impeded. It is our belief that a higher percentage of these cases are relieved if dilatation is practiced.

(11) *Cholangitis.* Varying degrees of cholangitis are met in many of the operations on the biliary tract. One may find the ducts so contracted at the time of operation that the supraduodenal portion of the common duct may be only a few millimeters in diameter. In such cases, it is difficult to safely explore the ducts. Stones may even be removed and the papilla dilated to the size of the duct only to have the patient's symptoms continue. At a secondary operation a few weeks later, the duct will be found dilated to sufficient size to satisfactorily remove other stones and dilate the outlet of the duct sufficiently to afford permanent relief. When one finds the duct lumen of good size at the time of the first operation, dilatation can be carried out sufficiently to make a secondary operation unlikely.

The most striking success was evidenced in a man of fifty-eight who had had his gall bladder removed twenty years before. He came to the hospital with symptoms of severe pain, nausea and vomiting of thirty-six hours' duration. His temperature and leucocytes were elevated and he was locally tender in his right upper quadrant. At operation his common duct was surrounded with an inflammatory exudate, there was no pancreatic thickening nor fat necrosis. The duct was opened with the discharge of thick yellowish-white material resembling pus. From this were cultured colon bacilli. No stone was found but the outlet of the duct was constricted. This was dilated to the size of the duct, No. 10 Bakes. A small catheter was placed within the duct and the opening sutured around it. The patient

became moderately jaundiced which completely cleared in seven days. His temperature gradually fell to normal and he left the hospital in good condition three weeks after admission. We believe that drainage of the duct to the outside alone might have relieved this patient, but are doubtful that recovery would have taken place by drainage alone as his story of indigestion for several months prior to entry makes us feel that there was a true constriction at the papilla and not one based purely on inflammation.

(12) *Impaired Liver Function.* Although one hesitates to subject a patient with marked dye retention to a serious surgical procedure, there are instances when this is necessary in order to restore him to health. Impairment of bile drainage will bring about liver damage in varying degrees dependent upon the amount of interference and the duration of time involved. One may assume that any patient with biliary tract disease based on stones, inflammation or previous surgery who has increased dye retention, will have some mechanical interference with bile drainage into the intestine. Common duct exploration with removal of stones in such instances is extremely gratifying. In some cases a dilated duct is found without stone and in these the papilla will be found constricted. It seems logical to us that liver damage will be corrected in a shorter period of time if internal drainage is carried out, than by means of prolonged external drainage. Bile is such an essential element to health that a more rapid recovery of the liver cells should take place if this is returned to the duodenum rather than to the outside.

In one instance a woman, who came in with a poorly functioning biliary fistula following cholecystectomy seven months before, had a 60 per cent dye retention. A stone was removed from the distal end of the common duct. The ampulla was dilated to the size of the duct, No. 10 Bakes, and a small catheter placed within the duct which drained the usual daily amount of between 300 and 400 c.c. after the seventh day. The catheter was removed on the tenth day with practically no further bile leakage. Much to our surprise her liver function was normal on the fourteenth day after operation. Doubtless this is an unusually rapid recovery and one would need to study many cases to determine the normal rate of functional return.

STATISTICAL DATA

In the period of time from January 1, 1931 to November 1, 1934, there have been 901 operations for biliary tract disease, exclusive of malignancy, at the Massachusetts General Hospital by members of the visiting and house staff. Fifty-seven of these were drainage operations only and represent the

TABLE 1
PRIMARY BILIARY TRACT OPERATIONS AND MORTALITY (1931–1934)
(Massachusetts General Hospital)

	1931		1932		1933		1934 (Incomplete)		Totals	
	Cases	*Deaths*	*Cases*	*Deaths*	*Cases*	*Deaths*	*Cases*	*Deaths*	*Cases*	*Deaths*
Cholecystostomy..	13	2	15	4	17	1	12	1	57	8
Cholecystectomy..	152	2	137	2	155	4	104	2	548	10
Cholecystectomy with common duct exploration and dilatation of sphincter of Oddi	13	0	33	2	39	0	34	2	119	4
Cholecystectomy with common duct exploration outlet not dilated	25	3	26	2	39	3	24	0	114	8
	203	7	211	10	250	8	174	5	838	30

acutely ill patients in poor condition. Of these, 8 died—a mortality of 14 per cent. Inasmuch as the majority of the staff feel that the gall bladder should be removed when possible even in the acute stage, we believe this group represents a more hazardous class than any, save those of prolonged obstructive jaundice. Primary cholecystectomy was done in 548 patients with 10 deaths—a mortality of less than 2 per cent, and among these are not only those with chronic disease but many in the acute and subacute stages without jaundice. In 269 common duct exploration was superimposed upon cholecystectomy. Of these, 131 did not have the sphincter

of Oddi dilated and 8 died; while 138 did have dilatation of the outlet averaging 7 mm. with 4 deaths. A few had very little dilatation and a few were carried up to 13 mm.

TABLE II
SECONDARY BILIARY TRACT OPERATIONS AND MORTALITY (1931–1934)
(Massachusetts General Hospital)

	1931		1932		1933		1934 (Incomplete)		Totals	
	Cases	*Deaths*	*Cases*	*Deaths*	*Cases*	*Deaths*	*Cases*	*Deaths*	*Cases*	*Deaths*
Cholecystectomy, previous cholecystostomy, common duct not dilated	5	1	4	0	14	1	4	0	27	2
Common duct exploration and dilatation of sphincter of Oddi	3	0	5	0	7	0	4	0	19	0
Common duct exploration, outlet not dilated	4	0	4	0	8	0	1	0	27	2
	12	1	13	0	29	1	9	0	63	2

TABLE III
TOTAL BILIARY TRACT OPERATIONS (1931–1934)
(Massachusetts General Hospital)

	Cases		*Deaths*		*Percentage*	
Cholecystostomy	57		8		14.0	
Cholecystectomy	548		10		1.8	
Cholecystectomy with common duct exploration and dilatation of sphincter of Oddi	138	269	4	12	2.9	4.5
Cholecystectomy with common duct exploration, outlet not dilated	131		8		6.1	
Cholecystectomy, previous cholecystostomy, common duct not explored	27		2		7.4	
	901		32		3.5	

There were 63 secondary operations on the biliary tract during this period. Twenty-seven had their previously drained

gall bladders removed. Seventeen had their common ducts explored and drained, and 19 had dilatation of the sphincter of Oddi in addition. Only 2 died in this secondary group and they had cholecystectomy only.

In the whole group of 901 biliary tract operations, 269 or approximately 30 per cent had the common duct explored. In 107 of these, stones were found, an incidence of stones in about 40 per cent of the ducts explored or approximately 12 per cent for the entire group.

TABLE IV

BILIARY TRACT OPERATIONS: CAUSES OF DEATH (1931–1934)
(Massachusetts General Hospital)

	Cases		*Cases*
Pneumonia	8	Bile peritonitis	2
Pulmonary emboli	7	Liver death	1
Hemorrhage	4	Myocardial failure	1
Pancreatitis	4	Shock	1
Peritonitis	3	Subdiaphragmatic abscess with rupture into pericardium	1

The mortality for the entire group was 3.5 per cent; for those with common duct exploration, 4.5 per cent.

POSSIBLE COMPLICATIONS AFTER DILATATION

One naturally approaches a new procedure with a great deal of care and thought as to the possible dangers that are involved. The advantages gained by such departures from previous methods must far outweigh these dangers if they exist. The first thought along this line was brought to our attention by Dr. Lincoln Davis[11] in raising the question of the possibility of duodenal reflex following dilatation of the papilla. Such complications have also been reported by Codman[12] and Walters.[13] In an analysis of Davis' 2 cases, we find that the common duct was "enormously" dilated at the time of operation and that although he ascertained, in one case at least, that the papilla was patent, he did not dilate it further. In these as well as Codman's and Walters' cases, the possibility of duodenal blockage beyond the papilla might have occurred. Codman felt that his case had obstruction

at Treitz' ligament by pressure from the mesenteric vessels. Actual erosion of the transduodenal portion of the duct may have taken place in these cases that would not have occurred had the outlet been well dilated. We were sufficiently impressed by the possibility of duodenal reflux if dilatation were done from our contact with Dr. Davis that we approached the question of mechanical dilators with a great deal of caution. In only one instance in our series of 138 dilatations, however, did we obtain any duodenal drainage and in this case, we feel sure that a false passage was made into the duodenum. This delayed the convalescence slightly but fortunately caused no serious damage. We feel now, as did Cheever and Bakeš, that duodenal reflux is not a likely or common sequel to carefully dilated papillae.

The possibility of flaring up an acute pancreatitis by dilatation of the transduodenal portion of the duct is one that naturally occurred to us. One patient did develop fatal acute hemorrhagic pancreatitis after cholecystectomy, common-duct exploration and dilatation of the papilla to 8 mm. The fact that the operator's note states that there was no evidence of pancreatitis at the time of operation with the immediate onset of vomiting, temperature, pain, tenderness and leucocytosis, finally resulting in death three weeks later, makes it necessary to accept this as a complication. It may be argued that one case of pancreatitis in 138 dilatations would represent a normal incidence of pancreatitis following any surgical procedure on the biliary system. As a matter of fact, there were only 4 deaths from pancreatitis in the 901 cases operated upon in this series. Heuer[14] states that in a very large group of collected reports that pancreatitis accounted for 2 per cent of the deaths.

The possibility of making a false passage through the wall of the duodenum into the peritoneal cavity has occurred to us. It seems evident that this technical error might take place. So far as we know, there has been no incidence in our cases. We still feel, however, that the danger exists and we believe

it can be avoided if the technique described is carefully followed.

When tissue is torn or injured, healing takes place with scar formation. We have, therefore, tried to evaluate the possibility of cicatricial constriction of the duct outlet after dilatation. There has, as yet, been no clinical evidence that this occurs. Certainly it is surprising that dilatation can be carried out with so little evidence of hemorrhagic reaction. If one carries out dilatation under direct vision with the duodenum open, the amount of trauma seems much less evident than in many of the cutting procedures commonly carried out for stones impacted in the ampulla, etc. We, therefore, doubt that constriction from scar tissue will develop following gentle, gradual dilatation.

DRAINAGE OF THE DUCT AFTER DILATATION OF THE PAPILLA

The question of drainage of the duct versus closure after dilatation of the papilla is a fair one. Bakeš believed that the hydraulic action of the closed duct was helpful in Nature's effort to evacuate stones from the duct after dilatation of the papilla. He admitted, however, that bile drained from the suture line to some extent in the majority of cases, although a certain amount of drainage may have come from the liver bed itself or from aberrant ducts. Cheever drained the duct routinely after dilatation of the papilla, recognizing the fact that tight closure is difficult and quoted cases from the literature of bile peritonitis when closure had been attempted even though the abdomen had been drained. One of the deaths in our series was doubtless due to the same cause. Also, 2 of our experimental animals died of bile peritonitis after the papilla had been dilated and the duct closed. It is probable that the instrumentation may create considerable secondary reaction in some instances, actually impeding the flow of bile into the intestine temporarily. We would like the hydraulic action in distending the duct and thus floating debris and stones through the outlet and this may have been effective in McKit-

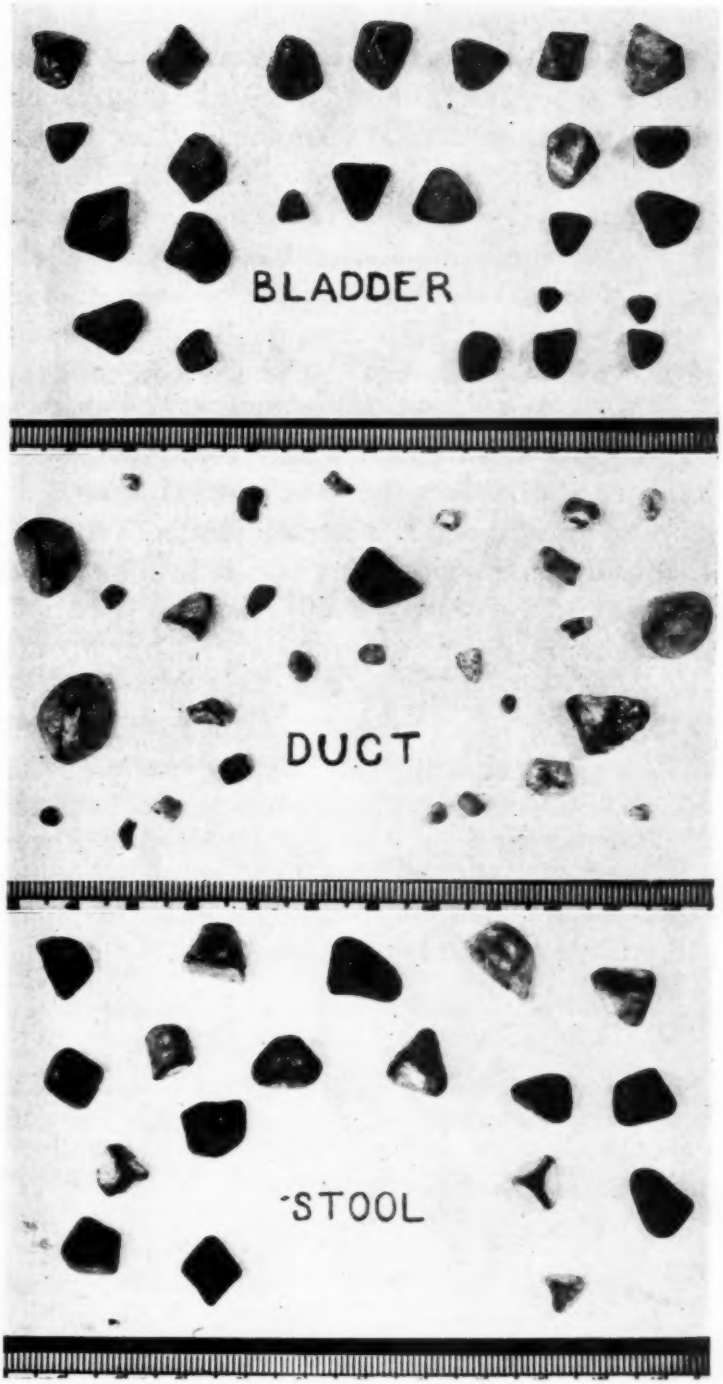

FIG. 7. Case of McKittrick's showing stones removed from gall bladder, from duct and collected in stool after dilatation of papilla to approximately 13 mm.

trick's[15] case (Fig. 7). We believe, however, that these stones would have passed through the 13 mm. dilatation whether the duct had been closed or not. It seems likely that in very large ducts where it is thought wise to dilate the outlet to a diameter approaching the size of the duct that closure is safe, provided care is taken to lead the bile leakage to the outside. We believe it is difficult to drain a profuse flow of bile along cigarette wicks even if they are placed in the subhepatic fossa. On the whole, we feel that there is not enough gained by attempting closure of the duct to warrant it, as a routine procedure. The small catheter in the duct doubtless relieves the pressure within the hepatic system and this we believe is a safety valve of importance and it may influence favorably the early repair of liver damage. Any reaction in the dilated outlet will have subsided by the time the catheter is removed and then the maximum benefit of the instrumentation will take place.

EXPERIMENTAL EVIDENCE

Large dogs were selected for the experiments. It was hoped to ascertain what effect dilatation of the papilla of Vater would have on normal dogs: (a) with the gall bladder intact; (b) with the gall bladder taken out at the time of original operation; (c) with a choledochostomy taking place two or three days after cholecystectomy.

The common duct in the dog is found to be quite thin-walled and difficult to suture tightly without constriction. It did not seem feasible to drain the abdominal cavity. Two of the animals died of bile peritonitis six and eleven days post-operative. The papilla had been dilated through an opening in the supraduodenal portion of the common duct. The incision in the duct was sutured with interrupted fine silk. Three dogs survived, as follows:

1. A male, weighing 24 kg. was operated on under nembutal anesthesia. The gall bladder was left untouched. The common duct was opened in the

supraduodenal portion and found to measure approximately 5 mm. in diameter. A 3 mm. probe could be passed through the papilla without impingement. A 4 mm. probe gripped firmly and a 5 mm. probe passed with considerable resistance. The duct was closed with interrupted silk stitches. The dog made a good convalescence and 118 days later weighed 38 kg. He was given chloroform intravenously. The region of the operative field was examined immediately. The common duct had a normal appearance. It was again 5 mm. in diameter. The duodenum was opened and the papilla looked entirely normal in size and shape. The 3 mm. dilator passed through without resistance. The 4 mm. dilator was slightly gripped and the 5 mm. passed about as readily as the 4 mm. dilator had at the original operation. There was no evidence of scar tissue.

2. A female weighing 27.6 kg. was operated upon under nembutal anesthesia. Cholecystectomy was done. Two days later nembutal anesthesia was again administered and the wound was reopened. The common duct was opened and was found to measure approximately 5 mm. in diameter. The 3 mm. dilator passed through the papilla without gripping. The 4 mm. gripped slightly. The 5 mm. dilator went through without obvious trauma. The dog survived and fifty days after the first operation gave birth to puppies. Seventy days after operation the dog was killed with intravenous chloroform. The common duct was found to be dilated to 8 mm. The duodenum was opened and the papilla had a normal appearance except that it was larger than at the first operation. The 3 and 4 mm. dilators went through without gripping. The 5 mm. dilator barely gripped. The 6 mm. dilator gripped about as much as the 5 mm. had at the original operation. There was very little evidence of scar tissue. The duct had healed so well that the previous incision could not be recognized. The walls of the duct, however, did seem a bit thicker than normal.

3. A male weighing 21.4 kg. had a cholecystectomy under nembutal anesthesia. Five days later the wound was reopened. The duodenum was opened over the papilla which would not admit the probe. Due to inflammatory reaction around the duct, it was not opened. The duodenum was closed. One hundred five days later the dog had gained to 32 kg. Under nembutal anesthesia a sterile operation was done. The common duct was found definitely larger than before, measuring approximately 7 mm. in diameter. The duodenum was again opened. The papilla was as normal in appearance as after cholecystectomy. The 3 mm. dilator passed without gripping. The 4 mm. dilator gripped slightly. The 5 mm. gripped about as firmly as other uninstrumentated papilla after delayed cholecystectomy. The duct outlet was stretched to approximately 7 mm. and the duodenum again closed, the scar tissue of the previous duodenotomy making closure a little difficult. If this dog survives we will attempt to determine how

much of the dilatation persists, as this represents the only pathological duct at our disposal.

Comment. 1. It would seem that the papilla of Vater in dogs can be dilated to the size of the supraduodenal portion of the common duct with safety. If this is done transduodenally there will be no bile leakage and it is a much safer procedure than dilatation through an opening in the duct, as external drainage in these animals does not seem feasible. It is difficult to tell whether or not bile peritonitis could be avoided by the usual drainage methods used in human beings.

2. The papilla in dogs is apparently somewhat more relaxed a few days after cholecystectomy than it is with the gall bladder intact or immediately after cholecystectomy. This coincides with Puestow's[16] experiments.

3. The common duct in dogs dilates after cholecystectomy as has been previously shown.

4. The dilatation of the normal papilla in dogs is not maintained up to the size originally dilated but apparently a second dilatation is carried out with greater ease.

5. Whether dilatation of the papilla to the size of a dilated duct which occurs following cholecystectomy will be maintained better than the normal papilla is yet to be determined.

6. Gross appearances do not show any evidence of trauma or scar tissue after healing has taken place. Microscopical sections were made on one case shortly after dilatation and these show some infiltration of red blood cells in the tissues but no serious hemorrhagic reaction.

SUMMARY

1. The indications for exploration of the common bile duct have been given.

2. Routine dilatation of the papilla of Vater in common-duct surgery and temporary external drainage of the duct by catheter is advocated.

3. A satisfactory surgical technique is given in detail.

4. Some experimental and considerable clinical evidence is given that dilatation of the papilla of Vater can be carried out with safety.

5. It is probable that dilatation of the outlet of the pathologically distended common bile duct may last some time.

6. It would appear that dilatation of the normal papilla in dogs is not permanent.

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[For Discussion see p. 569.]

STRICTURE OF THE COMMON BILE DUCT

REPORT OF A CASE

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THE literature of obstruction of the common bile duct has ceased to be frequent in recent publications. The treatment of such conditions has become so standardized that few surgeons report illustrative cases. During the first twenty years of the present century, there were many reports. A review of the publications of that period is replete with several different methods of treatment of such conditions. Probably, the reason for the absence of such reports is that the condition is infrequently met with because the surgery of the biliary tract and the gall bladder is much better understood and the patients come to the surgeon at a much earlier date than formerly and stricture of the common duct is not met with with the same frequency as during the earlier period.

In my own practice, it is quite rare to have charge of a patient suffering from long delayed gall-bladder pathology. The family physicians, who refer patients to me, diagnose their cases of gall-bladder and bile-duct disease at a much earlier stage of the disease than formerly. The general understanding of the medical ailments of the liver, gall bladder, and bile ducts has increased to a very great extent which accounts for the earlier diagnoses of any surgical condition which might terminate from medical diseases. Physicians have learned how to treat these ailments without leaving them to the fortunes of chance, later to end in some surgical condition necessitating operative measures.

There is nothing more interesting than the history of the development of the surgical understanding and treatment of

any specific disease. At first, the surgeon has no precept or guide to direct him and he must find the best method by the method of "trial and error" as all other pioneers have had to do. Often the emergency comes as a surprise and there is not time to study out the condition and to find the best way; the surgeon does the very best he can do under the circumstances. The situation is wholly different now because there is always a vast literature which every one who wishes may consult and find out what has been done in similar conditions.

REPORT OF A CASE OF STRICTURE OF THE COMMON DUCT

A woman, forty-nine years of age, came into the hospital on June 7, 1915, suffering from gall-bladder disease which she had had for more than twenty years; she had had many acute attacks during which she had fever, jaundice, nausea, vomiting, and typical pain. These spells came with irregular frequency, getting closer together and more severe until she was never free from pain and discomfort.

Her condition was very serious and all that could be done with safety was to drain the gall bladder, which was a small fibrotic, thick-walled mass, containing no stones; it was surrounded by very thick and strong adhesions to every adjacent viscus. She did well following the operation.

Soon after leaving the hospital she began to suffer as before the operation. She returned, and on August 25 the gall bladder was removed; the convalescence was normal and she did unusually well until in June, 1922, when she returned with all the symptoms of obstruction of the common duct.

At this operation, the entire region of the bile ducts and gall bladder was fibrous mass and it was with great difficulty that the common duct could be recognized. At the junction of the cystic and common ducts was a stricture almost completely closing the duct for about one inch. The stricture was excised and an end-to-end anastomosis was done. The results were satisfactory and the convalescence was normal for about two weeks. She then began to suffer as previously with all the usual symptoms of common-duct obstruction.

At operation on July 19, 1922, the entire common duct was found to be a hard fibrous cord and greatly enlarged; the previous operation was found to be intact and the healing was perfect. The duct was slit longitudinally and the lumen was barely large enough to admit a horse hair. The problem was: What could be done? There was no gall bladder; the duodenum could not be mobilized on account of adhesions. The entire

duct was excised, that is the part which was strictured. This left a section of the duct at the junction of the hepatic ducts about ¼ inch long and the duodenal end was so short that it was scarcely recognizable as a duct. In the emergency of the situation, it was decided to use a rubber tube from the hepatic ducts into the duodenum. A No. 16 F. catheter was split at one end for about ½ inch and each half was inserted into the hepatic ducts and the small segment of the common duct remaining was tied around it; the catheter was then laid in the channel of the common duct left after its removal and the other end of the catheter was passed into the duodenum for about one inch; the short segment of the common duct was tied around the catheter and then the tissues were carefully sutured over the catheter. A soft rubber drain was placed down to the gall-bladder region, but not to the rubber tube, and the wound closed.

The patient had a rather stormy convalescence for the first few days. After this, she did quite well. The drain was removed at the end of the second week and the wound healed. After going home she did well for three weeks and then a fistula developed at the upper end of the abdominal wound and discharged a bile-stained fluid for about two weeks and then it closed. She has done well ever since.

COMMENTS

This patient has had fairly good health all the time since the operation, although she is a confirmed morphine addict. She has been troubled with some occasional discomfort in the liver region, but, at no time has she had any symptoms of interference with the flow of bile into the duodenum; her skin has been slightly jaundiced on several occasions. It is now more than twelve years since the rubber tube was used to reconstruct the common bile duct and the patient has had no evidence that the bile is not passing into the duodenum. Later *x*-ray studies show that the catheter was passed through the intestines.

ALTERNATIVE PROCEDURES

An external fistula could have been made and later this might have been freed down to the duct and then inserted into the duodenum; this has been done with success. The hepatic

portion of the common duct was too short to insert into the duodenum as the duodenum could not be mobilized on account of the density of the adhesions around it. The hepatic duct could not be connected with the transverse colon for the same reason. So, there was nothing else to do but to establish an external fistula or to do what was done. The results seem to justify the use of the catheter.

The study of the various methods of reconstruction of the common bile duct dates back to the beginning of the early years of the present century. The problem had, undoubtedly, confronted many surgeons before their attempts at reconstruction were made public in the literature. The earliest cases were allowed to develop an external fistula and thereafter live a most miserable life. I am sure that many attempts were made to reform the bile ducts which were failures until some enterprising surgeon, bold in conception and courageous in act, decided to imitate the practice of urologists in restoring a section of destroyed urethra. The analogy, here, is not perfect as any possible resulting stricture of the urethra could be dilated, whereas a stricture of the newly formed bile duct could not be dilated. A hint as to what would happen to the new bile duct was given when observing an external fistula from the bile duct. The urethra is surrounded by a tissue which tends to scar formation to a remarkable degree, whereas the tissues surrounding the newly formed bile duct do not possess that characteristic.

There is, also, another quite different situation in the function of the two ducts, viz: the function of the urethra is irregular and occasional, while the common bile duct is functioning almost constantly with periods, such as after the ingestion of foods, of high pressure flow of bile. The more or less constant inside pressure within the common bile duct with periods of high pressure may have a decided relation to the prevention of stricture formation. Of course, this is a matter of mere theory based upon a quasi-physiological explanation. The fact remains, however, that the tendency

to stricture formation in the common bile duct is much less than in the urethra. The many successes of permanent function, following the use of a rubber tube in short resections of the bile duct, seem to lend support to the theory that the tendency to stricture formation is less than in the urethra. The bile duct is not, so far as is known, subject to localized acute bacterial infections which so frequently affect the urethra and produce destruction of the urethral wall resulting in fibrous tissue formation and narrowing of the lumen of the urethra. The infections of the bile duct are less severe and generally follow traumatisms from stones lodged in the duct. External infections may extend into the wall of the duct and cause tissue destruction with resultant fibrous tissue formation and constriction of the lumen.

The tendency to fibrous tissue formation in the tissues which reform the duct is lessened by the absence of muscle and fibrous layers which surround the urethra; also, the spongy tissues surrounding the urethra produce fibrous tissue as a healing process. This tendency is absent in the tissues which surround the reformed bile duct. The new duct is similar to and analogous with the fistula which forms the bile channel through the muscle walls of the abdomen.

LITERATURE ON RECONSTRUCTION OF THE COMMON BILE DUCT

The first reference that I have found bearing directly upon the reconstruction of the common bile duct is an article by Sullivan[1] published in 1909. In this article he describes a method which is very simple and, in his hands on experimental operations on dogs, quite successful. He uses a rubber tube to replace the gap in the duct; the tube is inserted into the upper end of the duct and then passes down to the duodenal wall and is inserted into the duodenum by a slit which is sutured over the tube for a few centimeters; a sponge is inserted into the duodenum, through an opening and tied or sutured to the tube. The object of the sponge is to draw the tube out of the

duct after the catgut sutures, which are used to fasten the tube to the intestine, have been absorbed.

Brewer,[2] in 1912, reported successful cases with Sullivan's technique; later, Arthur T. Mann[3] (1914) used the same technique, but tried to form an ampulla by pulling up the duodenal wall over the tube. He does not give his results with this added effort to make a more perfect common duct. In 1916, Riggs[4] wrote a very interesting paper in which he gives many different methods of repair of the obliterated common duct, one of which was by the use of an inlaid rubber tube. He quotes from a paper by Jacobson who gives the results of 34 cases of duct obliteration with various methods of repair of which 2 cases were with a rubber tube. He does not give details of the operations nor the technique employed. Again, in 1916, W. J. Mayo[5] reports cases in which the rubber tube was employed; he advises that the tube be extended into the duodenum for about one inch; his opinion is that this is the operation of choice and reports several cases treated with success by this method. In 1917, Ginsberg and Speese[6] report in detail a case in which, after failure by other methods, a rubber tube was used and covered over with autogenous omental graft with a well patient one year later. In 1918, Horsley[7] reported experimental cases in dogs in which a section of the jugular vein was used to fill the gap in the duct, but this method was unsuccessful. In the same year, Guerry[8] reported a further series of cases in which the tube was used with success. His case was, however, merely a small section of the duct which had to be repaired about ½ inch. In the discussion of Guerry's paper, McArthur[9] detailed 4 cases in which he had used a tube to bridge the gap in the duct; he advised that 6–8 inches of the tube should extend into the intestine. Ellsworth Eliot,[10] in 1918, reported cases in which the tube was used and left to itself to come down the intestine later. It is not necessary to give further reports here as the method, by this time, had been used by a number of surgeons and their results had been reported.

In al' of these reported cases there is very little said about the restoration of an entire duct. There have been many cases in which part of the duct was repaired with a tube into the duodenum. Sullivan's method and technique were generally used with modifications to meet the situation. My reading has not been extensive, but there is no reference that I have been able to find which reports the restoration of an entire common duct by the use of a rubber tube when the duodenum could not be made to approximate the upper end of the duct, or, at least, to shorten the distance between the two ends. In my case the distance between the liver end of the duct and the duodenum was more than 2 inches. Fibrous tissue formation from previous operations, and the peculiar pathology present, prevented any approximation to either end of the severed duct. The duodenal end of the duct was about ⅛ inch long after the most careful dissection of the duct was made; so, it was thought best to utilize this small segment of duct rather than to attempt to make an intestinal opening for the tube. It was dilated and the tube was passed into the duodenum easily. The postoperative history was free from any notable complications.

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DISCUSSION ON PAPERS OF DRS. ALLEN AND AYNESWORTH

Dr. Charles G. Mixter, Boston, Mass.: Residual obstruction of the common duct is one of the bugbears of surgery of that region. For a number of years at the Beth Israel Hospital we have studied the common bile duct radiographically after operation. During the past year an effort has been made to diminish the number of residual obstructions, and to determine which common duct should be opened. By the use of an opaque medium we have perfected a method of studying the bile ducts at the time of operation, which I think may be ultimately of value. When the common duct is exposed at operation a needle is introduced and bile is withdrawn for study, then 20 to 25 c.c. of an opaque solution (hipperan) are introduced and immediate roentgenograms taken on the table. It takes about six minutes to have the plates brought back for study. If we find dilatation we know that duct should be opened. After the duct is explored and the cause of obstruction, such as stone, has been removed, we put in a T-tube through which more of the solution is injected, and in another six minutes the plates are brought back. If the obstruction has been completely removed as shown by a free flow of medium into the duodenum, we feel that we can take out the gall bladder without danger of a residual biliary fistula. Any adjunct of this sort that will help the surgeon in demonstrating patency is certainly useful, and it gives me confidence, when I see the injection flowing freely through the ampulla into the duodenum, that the obstruction has been relieved.

A second point I might speak of is a little instrument that was made for me, a little soft silver catheter with a bougie point and a soft olive point fused on above the tip. This can be used for exploration. One can feel it as it goes through the ampulla into the duodenum, and one feels certain that it has passed into the intestine as fluid injected will not flow back through the common duct. It has proved very satisfactory to me.

Dr. J. S. Horsley, Richmond, Va.: Dr. Allen showed the necessity of dilating the common duct in many instances. I have used the duct stone forceps, introducing it through the duct into the duodenum and stretching it. Dr. Allen has a much more elegant technique, but the forceps seem effective and simpler.

Dr. Aynesworth is to be congratulated on meeting his emergency so effectively. Several years ago I was interested in reconstruction of the common duct and learned many things not to do.

I conceived the idea that the best method of reconstructing the common duct from the mechanical standpoint would be to use a vein. It is thin and by turning it inside out there is a surface that is comparable to the peritoneum, and it would be easily nourished. This idea of using a vein was not

original. We connected it with the end of the common duct in experimental work on dogs. The common duct in the dog has many branches. The vein was brought obliquely into the bowel, and was protected by suturing omentum around it. I thought this was a good operation. Later we put a little tube inside the vein in order to promote drainage so that the tube would finally be expelled. This was connected with a little thread which would somewhat guide it and cause it to be expelled. We kept these dogs for some time. Some died as the result of operation and the longest case was six or seven months old. The animals gradually died from obstruction of the duct. The point is that the vein is not accustomed to the bile. It is a question of biologic resistance. The soft mucosa of the stomach for instance is accustomed to gastric juice, but if gastric juice is placed on the skin, the skin ulcerates readily. It is a matter of tissue accommodating itself to its surroundings.

I think in Dr. Aynesworth's cases he used, perhaps unconsciously, this principle for the tissues around the common duct which are more accustomed to bile than a vein is, and there would be less tendency for contraction.

Dr. Frank H. Lahey, Boston, Mass.: I think we have been in the situation regarding gallstones that the urologists used to be in. I have used this comparison repeatedly. Urologists viewed the cause of mortality of prostatectomy for a number of years as being in the bladder, and it was not until they comprehended that it was in the kidney that they made any progress. We have viewed the mortality in gall-bladder disease as due to trouble in the gall bladder, but it is in reality largely in the liver.

Our figures of the incidence of common-duct stones found at operation which Dr. Allen quoted have crept from 8 per cent in 1926 to 21 per cent found and demonstrated at operation last year. I take a great deal of comfort from the autopsy figures of Krump. In 1000 consecutive autopsies he demonstrated stones in 380 instances, and in the common duct in 24 per cent of the cases. I think we have to admit that there are more common-duct stones than we formerly realized and that we have to open the common duct and look for stones more often if we do not want to overlook them.

I think if you realize that in all the cases in which we have found common-duct stones, jaundice was absent in 37 per cent of the cases, you will realize that we cannot depend upon jaundice as a sign of common-duct stones.

I have been interested in the fact that Dr. Allen's mortality is not high, even though they do a high percentage of explorations. Our own mortality has been reduced from 5.7 per cent to 1.7 per cent in spite of the increased percentage of ducts explored. You must remember that even if you take the gall bladder out if you leave a common-duct stone you

have probably left the stone which is producing the symptoms and certainly the one most apt to cause a fatality. It is a mistake to think that exploring the common duct will increase the mortality. It will in the hands of the inexperienced but not in the hands of the experienced surgeon.

We feel very strongly that this rule is true, that if the duct be not dilated you can remove the T-tube in twelve to fourteen days, but if the duct is widely dilated these patients should have the tube kept in for at least two months. The pathology is not only in the common duct but in the biliary tree, and there is a marked dilatation of the biliary tree. These patients need longer drainage, just as do patients with prostatic obstruction and kidney back pressure.

As relates to the use of a rubber tube in an operative defect in the common or hepatic duct, I think we must admit that practically all the measures of inserting of rubber tubes in place of the duct are usually unsatisfactory. The best method, when it can be done, of restoring the bile into the duodenum when the duct has been cut is by suturing the cut end of the duct into the duodenum.

Dr. John H. Lyons, Washington, D. C.: I wish to relate a case that I think is of interest in relation to Dr. Aynesworth's case. This was a patient of Dr. Mitchell's, of Washington, who was first seen in March, 1928, and who stated that a cholecystectomy had been performed four years previously. Shortly afterward he began to have attacks of severe pain in the upper abdomen, running through to the back and the right shoulder, and he also developed jaundice. He struggled on in this way for three years, when he was again operated upon and at that time a large number of adhesions were separated. He continued to have clay-colored stools, jaundice and attacks of pain.

Dr. Mitchell operated upon him within a few days after seeing him and found dense adhesions everywhere. No trace of the common duct could be found, but up near the liver the stump of the hepatic duct could be identified. The duodenum was mobilized so that it could be brought up against the stump of the hepatic duct. This was opened and a probe could be passed into it. A small opening was then made in the duodenum and a catheter passed into the hepatic duct and the other end into the duodenum. The tube was anchored in the hepatic duct, and the duodenum sutured to the stump of the hepatic duct. It practically amounted to suturing the duodenum to the liver. The patient made a good convalescence and within two weeks the jaundice disappeared. There was very little drainage and an *x*-ray picture showed the tube in the right side of the upper abdomen. He left the hospital in three weeks. In December, 1930, he again reported to Dr. Mitchell and said he had begun to get a little jaundiced and that his stools were getting a little lighter. The jaundice was not nearly so

marked as when he was first seen. A roentgenogram was made and showed the tube still present in the right upper quadrant, but it looked more feathery than at the time of the first *x*-ray examination, so it was felt that it was probably encrusted with salt and that its removal would help the patient. Dr. Mitchell operated again. At the first operation the duodenum was practical sutured to the liver. At this second operation it was about 3 cm. from the liver. In other words, a new common duct had been formed that was between 3 and 4 cm. long. Dr. Mitchell made an incision in this new common duct, removed the tube, closed the common duct, drained down to it and closed the abdomen. The patient left the hospital in a little more than two weeks and the jaundice rapidly cleared up.

This man was seen about two weeks ago, four years after the tube was removed, and he stated that he had experienced no trouble since the removal of the tube.

Dr. J. Tate Mason, Seattle, Wash.: Sir Berkeley Moynihan stated a good many years ago that secondary operations on the biliary tract are the most difficult in the realm of surgery and that to be successful one must have patience, craftsmanship, and a tranquility of mind that nothing can disturb.

I should like to present to you a method which we feel is very simple, first for anastomosing the gall bladder to the stomach and second for making an anastomosis between the common duct and the duodenum.

The gall bladder is first freed from its liver bed. A stab wound is then made in the greater curvature of the stomach just opposite the lesser curvature where the anastomosis between the fundus of the gall bladder and the stomach is to be made. We introduce a sharp-pointed hemostat, through the stab wound, and this can be pushed through the mucosa, the musculature, and the serosa from the inside of the stomach at the point where the anastomosis is to be made. Thus the muscles can be separated and not cut. Finally the hemostat is pushed through the serosa and the fundus of the gall bladder is clamped and pulled down into the stomach. One or two lines of sutures are then thrown around the line of anastomosis. The clamp is then released through the original stab wound in the lower end of the stomach, a small pair of scissors is introduced and the fundus of the gall bladder opened. Bile will immediately be seen to pour into the stomach. The operation is over, except for the abdominal closure, as soon as the stab wound in the greater curvature of the stomach is closed. This simple procedure not only saves time in the case of a patient who is usually very ill, but we feel has probably the advantage also of making a sphincter around the anastomosis because of the fact that the muscles of the stomach have been separated and not cut.

The second procedure which we feel offers a simplification is in the case of anastomosis between the common duct and the duodenum. The duct is exposed just above the duodenum and then two guy sutures placed on either side of it, which also catch a bit of the duodenum in with the sutures. A transverse incision is then made in the common duct and a stab wound opposite this in the duodenum. If possible and if it is not too difficult, one or two sutures are then placed in the posterior layer. A small rubber tube about 2 cm. long and the size of the common duct is then placed with one half in the common duct and the other half down into the duodenum. Before being placed in position a small silk suture is attached to the middle of the tube, which allows one to tug on the tube and hold the whole operative field up while it is closed tightly around. When the anastomosis has been sutured around as tightly as possible, this suture is cut at the line of anastomosis. By placing one finger above the upper end of the tube it can be partially pushed into the duodenum, leaving one-third of the length of the tube above the anastomosis, and thus the tube holds the lumen open a few hours until the tissues have become sealed over the suture line.

We feel that these two simple procedures have often shortened the period of operation by three-fourths of the time it would have taken by an ordinary suture anastomosis. I think that we must understand, though, that while many of these anastomoses are absolutely essential any time an anastomosis is made between any part of the biliary tract and a hollow viscus there is invariably an ascending infection, which usually begins as a mild hepatitis and may progress to a condition of multiple abscesses of the liver. Many of the older patients with carcinoma of the head of the pancreas will die from this ascending infection before long.

Dr. W. O. Floyd, Nashville, Tenn.: I report a successful recent case rather similar to that of Dr. Aynesworth, in which the patient had previously had a cholecystitis. The cholecystitis, chronic in character, had been followed by cholecystectomy about nine months before I saw her, at which time no stones were found in the gall bladder and none were palpated in the common duct. Owing to the fact that there were no stones in the gall bladder at the time of the cholecystectomy the duct was not explored. This patient had a rather stormy convalescence, with considerable nausea and vomiting. On the eighteenth day the wound broke open, requiring secondary closure. She left the hospital several weeks later. Soon after her return home she developed nausea and vomiting again, and soon noticed that she was becoming jaundiced. The jaundice increased in depth, and the nausea and vomiting increased to a point where she could retain but very little nourishment.

When I saw her, about eight months later, she had lost 25 or 30 pounds owing to the great amount of nausea, although she was very small naturally, weighing only 125 pounds. At this time she was deeply jaundiced and had a rash that completely covered her body. This dermatosis bled upon scratching. In addition to these symptoms she had had for the past three months a great many chills associated with a temperature that went as high as 103° or 105°F. I made a diagnosis of stone in the common duct that had been overlooked at the previous operation. The question of whether the jaundice had been disappearing or was clearer at times could not be determined from the family physician's report or the patient's statement. Upon exploration we found adjoining viscera densely adherent to the liver especially the duodenum, which we accidentally tore into on getting down to the duct structures. We could not identify the duct, and although the patient had a coagulation time of three and a half minutes and a bleeding time of one and a half minutes every torn vessel bled very freely. Aspirations of the duct tissues in an effort to obtain bile left profuse bleeding, despite the bleeding time and coagulation time. After prolonged effort we were able to identify what we thought was the remains of the common duct. Tracing it to the liver, we finally found profuse bile very close to the liver, so close we had no remains of the common duct to work with. Realizing that these cases may be lost from hemorrhage, we decided to make a temporary fistula to get rid of the jaundice before transplanting the duct. We used a large sized catheter in the hepatic duct, which was brought outside, put a drain in Morison's pouch and closed the abdomen. At that time she had a large hernia which we made no effort to repair. About ten days later, with the jaundice greatly improved and the patient able to retain nourishment, we reopened the abdomen, removed the catheter and drain. We inserted a No. 16 catheter into the intrahepatic duct and into the duodenum. The catheter was held in place by a silk suture through the duodenum after the incision in the duodenum had been closed with a purse string suture. The duodenum was then sutured to the liver around the catheter, there being insufficient duct remains to suture to. A Penrose drain was again placed in Morison's pouch, and the hernia repaired on closure of the wound. The patient did well, left the hospital eighteen days later and has remained well now for nearly a year.

UNUSUAL TUMOR (MALIGNANT ADENOMA) OF LIVER IN BABY

FLOYD W. MCRAE, M.D., F.A.C.S.

ATLANTA, GA.

THIS patient, a boy, six months of age, was brought to the Henrietta Egleston Hospital for Children by his mother, who had noticed an enlargement of the right side of the abdomen. This enlargement had been gradual over a period of about two and one-half months. There had been no other signs or symptoms.

History. The important positive findings were: The patient's birth was normal, as was his feeding. Father's age twenty-six, mother's twenty, both well. One brother three years of age, normal. There had been two miscarriages, second and third months, but no stillbirths. No history or evidence of tuberculosis, cancer or syphilis.

Examination. Patient appears normal except for slightly pale mucous membrane; cheerful and alert. Weight 19 lbs. 5 ozs. Physical findings grossly normal except for abdomen where there is noted a definite protusion in the right upper quadrant, extending from under the costal margin to the right anterior superior spine. It is more prominent when the patient is upright. There is no rigidity, no fluid or other masses. The mass is somewhat irregular in outline with suggested nodulations and areas of less resistance. It extends beyond the midline, and the liver cannot be differentiated. The tumor can be palpated posteriorly but one gets the impression that it is anterior to the kidney. No changes noted with respiration but the mass is movable.

Laboratory. There is an anemia. Red blood count of 3,550,000; 45 per cent hemoglobin. White blood count of 9400; 88 per cent polymorphonuclears. Urinalysis: Faint trace of albumin with an occasional finely granular cast. Wassermann and tuberculin tests negative. *X-ray* (Dr. J. J. Clark). Chest and skull negative. Neoskiodan given and both kidneys were found to be normal in size and shape; calices and pelves showed no deformity. Gastrointestinal Series: The stomach, colon and small intestines were displaced downward and to the left by the mass in the upper right quadrant which is in intimate contact with the liver and extends down from the right lobe. No pathology is noted in the stomach or intestines.

Impression. Patient was seen by various members of the Staff and after eliminating the kidneys and intestinal tract, tumor mass was thought to be: (1) lymphosarcoma, probably of mesenteric origin, or (2) tumor of

the liver. As a means of differential diagnosis deep *x*-ray therapy was given by Dr. J. J. Clark, and a three weeks' period was allowed to determine the effect. There being no noticeable change except a slight increase in anemia,

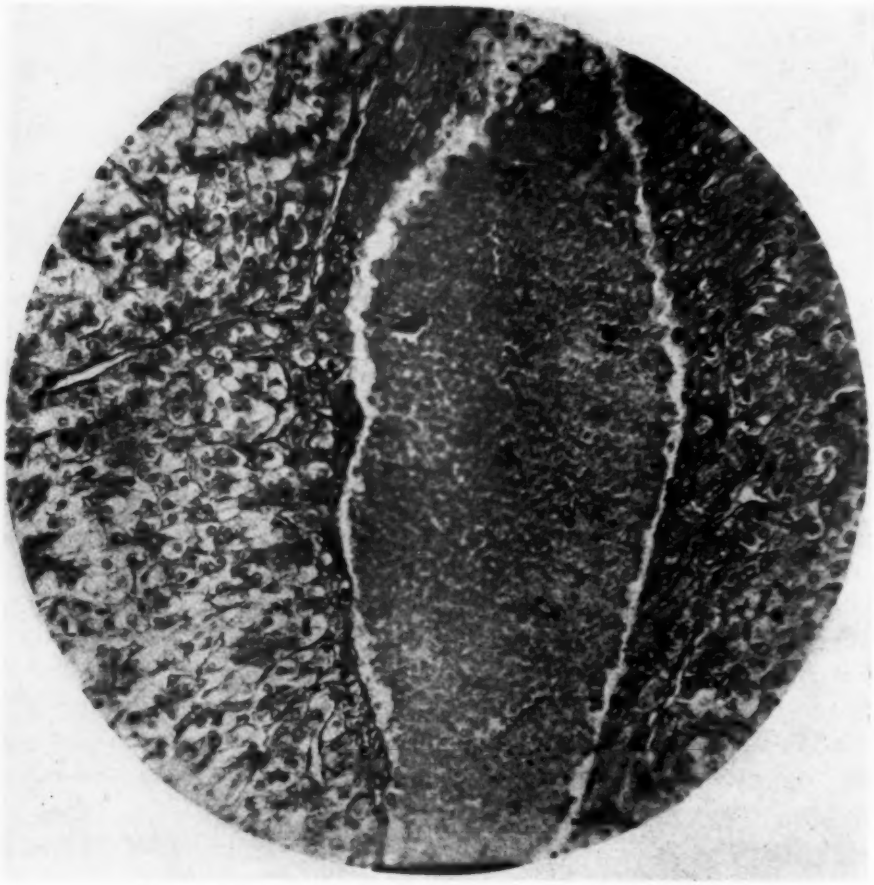

FIG. 1. Liver cells in cords and cavernous blood spaces.

patient returned to Hospital for operation.

Operation. The abdomen was opened through a right paramedian incision. There was no free fluid and the tumor was intimately attached to the liver, in fact appeared a part of the right lobe and no line of cleavage could be determined. The liver itself was smooth and no other nodules could be felt. No adhesions to the adjacent organs; no glanular enlargement. I was not sure whether the mass could be removed surgically, but realizing the hopelessness of the situation, believed that an attempt should be made. As far as possible, the blood vessels, which were quite numerous,

were divided between clamps, and I was able to excise the mass with sharp and blunt dissection with very little loss of blood. An attempt was made to close the raw surface of the liver with mattress sutures. A dry

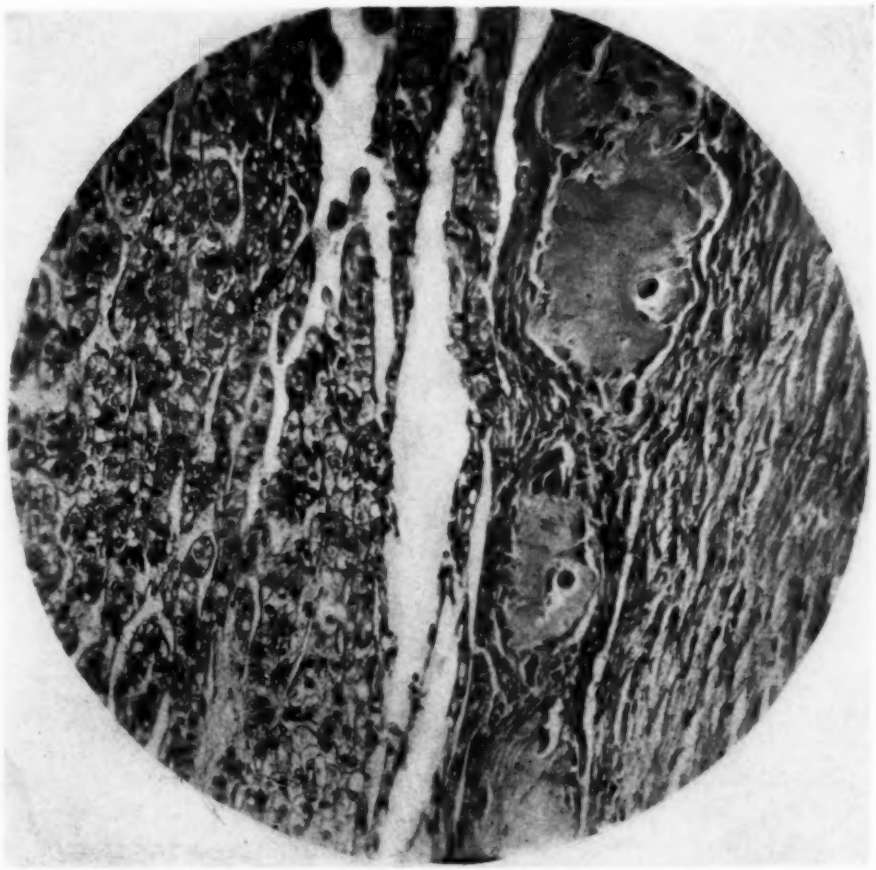

FIG. 2. Adenomatous structure with osteoid tissue in capsule.

gauze pack surrounded by vaseline gauze was placed against the surface of the liver, and the abdomen closed routinely; 50 c.c. of Amfetin being left in. The patient stood the operative procedure remarkably well, and was given 120 c.c. of his father's blood and glucose before leaving the operating room.

Postoperative Course. Condition was satisfactory with baby taking fluids without vomiting. There was no evidence of hemorrhage; abdomen soft, without distention. Temperature 103°F. Pulse 150–160. On the fourth postoperative day patient vomited rather profusely and looked sick. There was a stool but the abdomen was slightly distended. He was given a

second transfusion, glucose and gastric lavage. Flatus passed, as well as a small stool. The fifth day patient became somewhat cyanotic and there was an icteric tint to the conjunctivae; extremities cold and about two hours

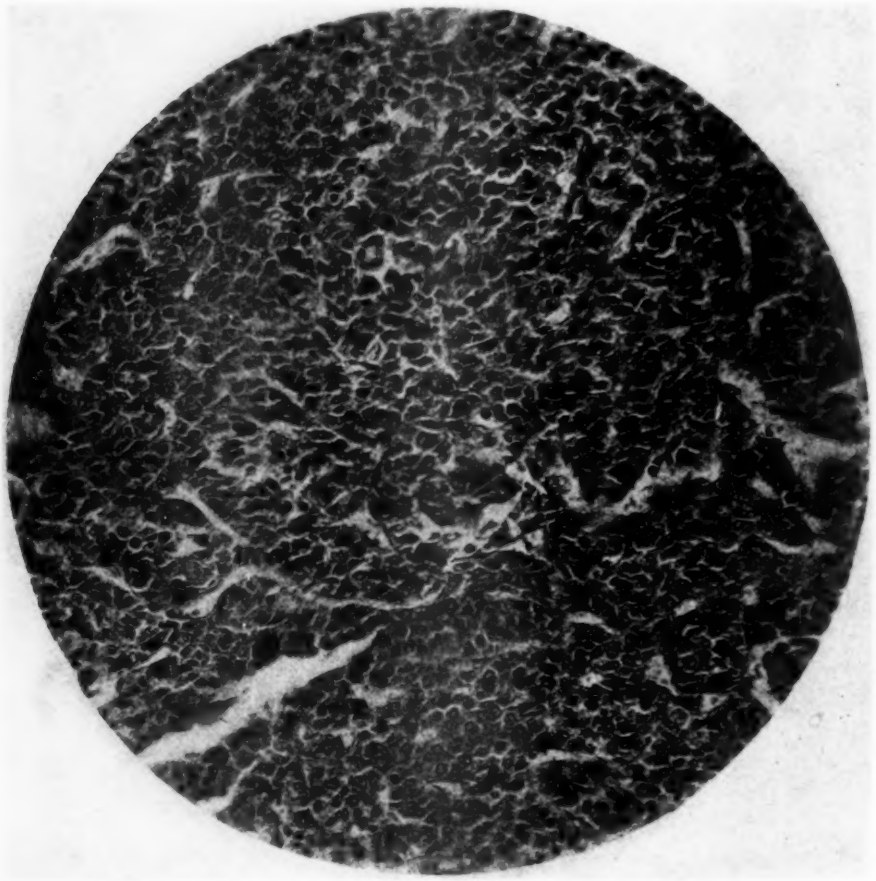

FIG. 3. Cellular active area of diffuse part of tumor, low power.

before death the vomitus was somewhat fecal in character. Abdomen was still soft. He died quite suddenly.

I felt that death was due to hepatic insufficiency and partial intestinal obstruction.

Autopsy. As the abdomen was opened a large amount of light yellow, clear, fluid was found. Small intestines were distended with several areas of discoloration. There was no evidence of mesenteric thrombosis. In right upper quadrant two gauze sponges were in contact with a loop of the ileum and thin recent adhesions had formed; this produced partial obstruc-

tion. There was nc gangrene. On the inferior surface of the right lobe of the liver there was an oval depression about 1.5 cm. deep. The central portion was denuded and several mattress sutures were in the liver substances.

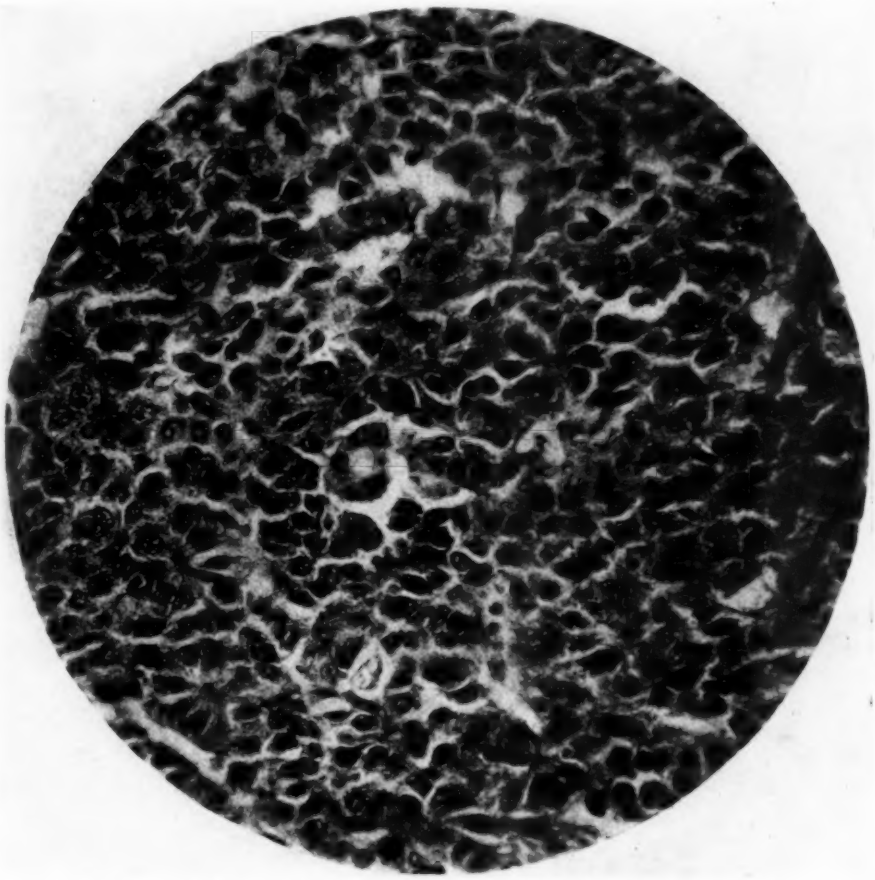

FIG. 4. Cellular active area of diffuse part of tumor, high power.

The flat gauze was adherent and there was no evidence of hemorrhage. A small gall bladder was distended with bile; there were no stones. No gross masses felt in the liver which was pale and smooth. Kidneys grossly presented no abnormality. The mesenteric lymph nodules were enlarged, none showing any evidence of metastases. Death was result of hepatic insufficiency and partial postoperative obstruction of the ileum due to adhesions about the gauze pack. (Dr. Jack C. Norris.)

Specimen: Gross: The specimen is that of an ovoid, semi-fluctuant, thinly encapsulated tumor which measures 13 × 8 × 6 cm. and weighs

322 gm. It is pinkish red in color and is irregular and nodular in shape. The irregularity of shape is the result of variably sized semi-resistant nodulations. Cut sections reveal the tumor to be separate in character and is composed of fairly well-divided lobular aggregations of different color and consistency. Some are of a light brown and others red from increased vascularity and areas of hemorrhage. The entire process is very friable. One gains the impression that the tumor is of a most uncommon type, probably of an adenomatous nature which is attempting to reproduce atypical liver lobules.

Microscopic: Sections from various portions of the tumor show quite a wide variety of structures. The tumor is definitely lobulated, the lobules being separated by moderately dense and fairly wide bands of connective tissue. In and adjacent to some of these connective tissue bands, there are masses of hyalinized and osteoid tissue. The bulk of the tumor is composed of large sheets of medium-sized hyperchromatic cells, which cells are arranged in cords, columns and alveoli. Many groups of cells, in fact *sheets* of cells, show marked vacuolization, resembling lipoid change in liver cells. In other portions the cells are smaller, with acidophilic and finely granular cytoplasm, and while they appear in sheets, close inspection shows the cord formation as seen elsewhere. The cells also form masses which are separated by sinusoids. There are numerous large vascular spaces among the tumor cells. These spaces do not have any well-developed lining, but the blood is in direct contact with the tumor cells. These blood spaces are found not only in the marked neoplastic areas but also among the more normal appearing hepatic cells. In one area there is a concentric mass which closely resembles fusion of pavement epithelium with masses of keratinized granules in some of the cells. Necrosis is present and varies in amount in different areas. Mitoses are absent in some portions, and fairly numerous in those areas composed of more atypical or embryonal cells.

Discussion: It is perfectly evident from the character of this tumor that it is primarily one of liver cells and while the bulk of the tumor presents a structure of an adenoma yet there are areas in which the tumor is more definitely malignant and therefore of carcinomatous character. The evidence of actual malignancy is also supported by the poorly formed vessels, the numerous mitoses in some portions, the embryonal character of the cells and the active invasion of the trabeculae and capsules by small groups of these cells. The numerous areas containing large vascular spaces suggest a cavernoma or hamartoma and it is possible that this tumor is in reality a congenital mixed tumor, primarily adenoma and adenocarcinoma, with areas of osteoid tissue, atypical blood vessels and a small area of keratinized epithelium contributing evidence of such a tumor of mixed character. (Dr. E. L. Bishop.)

SUMMARY

1. A case is presented of a congenital tumor of the liver in an infant which shows pathological changes indicative of adenoma, carcinoma and mixed tumor.

2. Deep *x*-ray therapy had been given without appreciable effect.

3. Surgical removal should always be undertaken as the tumor is not invasive and it is my opinion that recurrence will be very rare, providing patient survives surgical procedure.

DISCUSSION

DR. ALBERT O. SINGLETON, Galveston, Texas: My interest in Dr. McRae's subject was stimulated by a fairly similar case which was encountered about three years ago. The patient was a child that was born in the hospital, the delivery being normal and the infant weighing 6 pounds. Immediately after birth a large tumor was recognized in the right side in the region of the liver. There was no effort to determine whether it was from the liver or the kidney and we were never certain about this previous to operation. The kidney pelvis was outlined by skiodan but indefinitely, and an exploration was made when the infant was seventeen days old. A large tumor was found growing from the liver, that was quite similar in appearance to the one just presented. It was removed with the cautery, the abdomen closed and the child recovered satisfactorily and is now well, three years after operation.

The tumor weighed 175 gm. in an infant weighing 6 pounds. It was well encapsulated with areas of hemorrhage in various locations, and there was some effort at cyst formation. Some areas were found in which the epithelium was proliferated, and we were of the opinion that we were dealing with adenocarcinoma. We submitted the tissue to several pathologists and Dr. Ewing thought it was an adenocarcinoma, with a tendency to recur. He stated that these growths are rare.

SURGICAL EMERGENCIES DURING PREGNANCY

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THE period of pregnancy is certainly not the time of election for major surgical procedures, but the woman in the pregnant state is subject to the same surgical diseases which may call for intervention in the non-pregnant. The aim and objective in obstetrics is to furnish safe conduct through pregnancy for both the mother and the child. However, all acute surgical conditions should be dealt with in the same manner as in the non-pregnant woman and, although the pregnancy adds gravity to the situation, the problem should become primarily a surgical one. The fact that a pregnancy exists demands much more careful study and painstaking investigation. This is particularly true in the presence of abdominal symptoms. Abdominal pains are of frequent occurrence during pregnancy and should never be disregarded or passed over lightly. The clinical picture of an acute abdomen should always call for prompt and decisive action. Doctors doing obstetrical work, unless surgically minded, are naturally inclined to delay operation.

The necessity for operation during pregnancy need cause no undue concern. Hippocrates wrote that acute diseases of the pregnant woman were always mortal, but this is no longer true of acute surgical diseases. The improvements in technique and anesthesia together with proper regard for careful and gentle manipulation during operation and full attention to preparation and aftercare have greatly reduced the mortality and the chances of interruption of the pregnancy by operative measures, even those of major proportions. The incidence of abortion is surprisingly low. Abortions will occur but they are often the result of the disease and not of the operation. There

are numerous reports in the literature of gastrectomy, nephrectomy, resection of the colon, cholecystectomy etc. which have been survived by both mother and fetus. Mussey, at the Mayo Clinic, reported a few years ago a series of 370 operations upon pregnant women. The list included operations on the appendix, gall bladder, thyroid, breast, ovary, kidney, stomach, etc. There were 4 deaths and only 17 abortions. Similar reports have been made from other clinics.

Where the condition is acute the line of action is usually clear cut, but other complications arise which are open to debate as to whether operation is necessary or justified during pregnancy. Many factors must be taken into consideration, chief among these being the risk to the mother and the child. Is the risk greater with or without operation? If operation is elected the next consideration is the most favorable time. If there is no urgency it is best to wait until after the tenth or twelfth week or until after placentation is complete. After the fifth month abdominal operations should not be done unless absolutely necessary. Sometimes the problem involves the question as to the advisability of terminating the pregnancy as a preliminary to operation. This procedure may become necessary in the interests of the mother.

As would be expected, appendicitis is probably the most frequent acute surgical disease of pregnancy. Early recognition is of vital importance to the mother and the prospect of preserving gestation is always better when surgery is not delayed. The unfavorable prognosis of appendicitis in pregnancy is due to the fact that perforation and extension occur much earlier because the pregnant uterus may interfere with the usual protective walling-off process that so often prevents disaster in acute appendicitis. Such an acute process becomes more and more serious as pregnancy advances. In case of doubt it is always better to risk operation than to defer surgery at the possibility of still greater risk.

Early recognition is not always so simple even in the non-pregnant woman and preganancy adds to the difficulties. A

history of previous attacks is helpful and ruling out non-surgical conditions will serve to clarify the issue. Investigation of the urinary tract is important, but pus in the urine does not eliminate appendicitis. Differentiation from ectopic pregnancy and tumors with twisted pedicles is unimportant for these are also acute surgical emergencies.

Ovarian cysts or tumors coincident with pregnancy bring into the picture another condition which may complicate pregnancy and labor. When recognized early in the pregnancy it is usually best to operate because of the increased likelihood of twisted pedicle, the tendency to rapid growth and the possibility of rupture and infection. If ovarian tumors are not discovered until the later months of pregnancy it is probably best to wait and do a section at term and then remove the growth.

Most patients with fibroid tumors of the uterus will have little or no difficulty during pregnancy. These tumors should be watched and operation done if necessity arises. The main indications for surgery are interference with ascent of the uterus from the pelvis, rapid growth or excessive pain. The possibility of rupture of the uterus due to interstitial fibroids must be kept in mind. I have seen one such case where spontaneous rupture of the uterus occurred at about the fifth month in a patient with a fibroid uterus. On the other hand rupture might take place as the result of an operative scar in the uterus where a fibroid had been removed. The patient with fibroids must be individualized but as a rule these tumors are inconsequential and of little clinical significance. Women with fibroids are inclined to sterility and the fibroids are an etiological factor in abortion and miscarriage.

Many other surgical procedures can be safely carried out during pregnancy. The welfare and comfort of the mother often necessitates removal of hemorrhoids and this can usually be done with little risk. Thyroidectomy, breast operations, tonsillectomy, dental operations and so on may be done when the best interests of the mother and baby seem to demand

them. Mussey in his report from the Mayo clinic listed 55 thyroid operations with 3 abortions and no deaths. Frazier and Ulrich have recently reported 7 operations on the thyroid in pregnant women with no abortions and no deaths.

Cholecystitis should receive palliative treatment as a rule but there should be no hesitancy in operating on the biliary tract when necessary.

Pyelitis in the milder attacks should be treated medically but ureteral catheterization should be done promptly in the severe cases and a catheter left in the ureter on the affected side. This has given me excellent results in a number of instances.

In a series of about 2000 obstetrical patients I have found it necessary to do a laparotomy in 19. Five of these operations could be classed as acute emergencies, 8 as urgent elective operations and 6 as frankly elective. There were 3 abortions and no maternal deaths. The 3 abortions occurred in the urgent elective group. Operation for hemorrhoids has been performed a number of times and also operations on the breast. Many of the patients have had dental extractions and a few have had the tonsils removed. All these latter procedures have been without bad results.

Appendicitis has been encountered 9 times. In each instance the trouble developed in the first half of pregnancy and operation was attended by no untoward effects. One patient aborted some three weeks later and after she had been up and around. This abortion was not considered as an operative result for the patient had had one spontaneous abortion prior to this and has just recently had a hydatid mole. She had had one full-time pregnancy.

Four of the appendix cases were of the acute fulminating type and the other 5 were sub-acute.

Operation for fibroids of the uterus was done 4 times and abortion occurred twice. Both of these patients had severe pelvic pain which necessitated the operation and both of them have had two full-term pregnancies since. One of them had a

recurrence of fibroids and I did a cesarean section at term and removed the uterus. In 4 other patients the uterus was removed following cesarean section because of fibroids. The 2 patients who did not abort after myomectomy completed their pregnancies. One of them, a primipara, who had a large fibroid removed from the uterocervical junction of the uterus last March went through a very normal labor early in October. Numerous instances of innocuous fibroids have been seen. At times more or less pain was experienced by the patients but never severe enough to indicate operation.

Ovarian cysts of pathological proportions were found as complications in 6 patients. Operation was done during pregnancy on 4 of these with one abortion. The tumors were all simple cystomas. One of the patients, while shopping for some things in preparation for operation, had the cyst twist on its pedicle and had to be taken to the hospital for an emergency operation.

The fifth patient in this group had a very large multilocular cystadenoma. Influenced by the doctor with whom I saw this patient, I terminated the pregnancy and removed the cyst a few days later. I have always since felt that interruption of the pregnancy was not really necessary. The patient has had a baby since this experience.

With the sixth patient the cyst was not recognized until after delivery. I am reporting this case to show what may develop in ovarian cysts as a postpartum complication. The cyst was discovered high in the left abdomen shortly after labor was ended. It was apparently adherent and would not descend with the uterus. In a few days it became quite painful and tender and later became infected. Operation for incision and drainage was done by another surgeon five or six weeks postpartum.

SUMMARY

Whenever possible operation for conditions arising during pregnancy should be postponed until after delivery.

Acute abdominal conditions demand prompt surgical intervention and watchful waiting is never the safe policy.

Pregnant women with pelvic or abdominal tumors should receive such treatment as seems best in each individual case.

Various surgical measures can be carried out during pregnancy when the necessity arises with surprisingly little risk to the mother and child.

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[For Discussion see p. 596.]

TOTAL VERSUS SUBTOTAL ABDOMINAL HYSTERECTOMY IN BENIGN UTERINE DISEASE*

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FIVE years ago I presented before this Society my simplified technique for total removal of the uterus by the abdominal route and discussed the indications for the use of this more radical operation as distinguished from subtotal or supravaginal hysterectomy. At that time I expressed emphatic opposition to the routine employment of total hysterectomy in all cases and stated that it should be resorted to only when operable malignant disease of the body of the uterus is encountered or when, in addition to benign disease of the corpus requiring hysterectomy, it can be clearly demonstrated that menacing pathology of a benign nature coexists in the cervix.

It is generally understood, of course, that for a number of years this has been and still is a controversial subject and that the conservative opinion just expressed is by no means subscribed to by all those who have participated in the discussions. On the contrary, the multiplying reports within recent years of a surprisingly large number of cervical stump cancers has been largely responsible for the adoption by some eminent surgeons of routine total hysterectomy. And these same reports, I am frank to confess, unsettled my own convictions a bit and stimulated me to undertake a comprehensive review of the literature in order to evaluate impartially the accumulated factual as well as the inferential data pertinent to this controversy. It is my purpose to present here only a brief synopsis of this study in hopes that it may prove helpful in bringing about greater unanimity both of surgical teaching and practice as regards the preferential operative treatment of benign uterine maladies.

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Perhaps the most cogent argument offered in support of their contention by the advocates of routine total hysterectomy is the apparent steadily mounting incidence of cervical stump cancer. Judging from my own observations alone, extending now over a period of twenty-five years of active clinic work and private practice, I would unhesitatingly conclude that the occurrence of stump cancer is so rare as to be a negligible factor in this discussion. Consequently, great indeed was my surprise to learn from a recent publication by von Graff that he had been able to assemble nearly 1200 cases, two-thirds of which have been reported within the last twelve years. Certainly, therefore, cancer of the cervical stump can no longer be considered a rarity. Unfortunately, it is impossible to determine with any degree of accuracy its absolute incidence because no one has the remotest idea how many thousand subtotal hysterectomies have been performed the world over to produce 1200 instances of stump cancer. Nor, on the other hand, is it at all probable that this number represents the total incidence, since scattered cases have been observed here and there which have never been reported. The average incidence subsequent to approximately 10,000 subtotal hysterectomies reported by a dozen different authors is a little less than 1 per cent. But in a study at the Johns Hopkins Clinic just completed and not yet published Erle Henriksen found among 940 cases of cancer of the cervix an incidence of 2.3 per cent of stump cancer. In some other statistical reports of the same kind this percentage is considerably higher, the average being around 4 per cent. Curiously enough, in the literature on cervical stump cancer both from American and European sources one repeatedly encounters the testimony of men who for years have been either at the head of or closely identified with large and active gynecological clinics that they have never seen a single case of stump cancer. Such wide statistical variations are to be explained probably by the fact that within recent years most of the cervical cancer cases have been referred for treatment only to institutions which are adequately

equipped for irradiation therapy. All factors considered, I do not believe that the actual incidence of stump cancer the world over today exceeds 3 per cent.

In this connection it is important to consider the time interval between the performance of subtotal hysterectomy and the subsequent appearance of stump cancer. Most writers upon the subject have arbitrarily assumed that all cancers becoming manifest within one year were present and overlooked at the time of the operation. Now over 20 per cent of the cases fall into this category. The remainder, constituting more than three-fourths of the total incidence, have developed after periods of from one to twenty years and all of these, according to the claims of its advocates, would have been saved by routine total hysterectomy. Obviously this assertion is entirely unwarranted, since not only does it inaccurately assume that the more radical operation as performed by the average surgeon carries with it no mortality whatsoever, but also it utterly ignores authentic reports of a score or more of vaginal vault cancers which developed subsequent to total hysterectomy, thus proving conclusively that even this procedure does not afford absolute protection.

But the major fallacy in the argument of those who contend that the present incidence of stump cancer is itself reason sufficient for the universal adoption of routine total hysterectomy is the significant fact that absolutely no data are available for determining the condition at the time of the original operation of those cervices in which cancer later developed. It is pretty safe to assume that most of them occurred in cervices which at the time of the subtotal hysterectomy presented the familiar picture of old lacerations, hypertrophy, so-called erosion and chronic infection. Certainly it would be very easy to build up convincing circumstantial evidence in support of this assumption. And if this be true, the incidence of stump cancer should be construed not as a compelling reason for the universal adoption of routine total hysterectomy, but rather as an index of the unfortunate

choice of cases suitable for subtotal hysterectomy. This latter operation is properly applicable to only four types of cases: (1) to those women requiring hysterectomy for benign disease who possess perfectly normal cervices; (2) to cases where the operative hazard compels the execution of rapid and conservative surgery; (3) to a few cases where for good and sufficient reason it is of paramount importance to preserve the function of menstruation; and (4) to most cases requiring hysterectomy during pregnancy. Before deciding upon subtotal hysterectomy, therefore, it is incumbent upon the surgeon to scrutinize the cervix with particular care, making use not only of intelligent palpation and accurate inspection of it but also utilizing freely such diagnostic aids as the colposcope, the Schiller test, biopsy and diagnostic curettage. Furthermore, remembering how frequently coexisting carcinoma at and above the level of the internal os is overlooked, the body of the uterus immediately upon its removal by subtotal hysterectomy should be laid wide open so as to permit accurate inspection of every centimeter of the endometrium; likewise all fibroid tumors should be bisected and carefully examined and an immediate frozen section report should be obtained from any suspicious areas. There is scant likelihood that stump cancer will later develop in any cervix which survives the rigid application of these tests.

Much has been written concerning the predisposing influence upon the development of cancer of the uterus which appears to reside in fibroid tumors. Of course it has long been known that with at least 3 per cent of all fibroid tumors there coexists malignant disease in the form of adenocarcinoma of the uterine cavity, sarcomatous degeneration of the fibroid tumors themselves or cancer of the cervix. But recent statistical studies of uterine cancer reveal the striking fact that cancer of the body occurs nine times and cancer of the cervix four times oftener in association with fibroid tumors than is the incidence in otherwise normal uteri. Moreover, fully two-thirds of the cases of stump cancer thus far reported have

followed subtotal hysterectomy for fibroid tumors. Here it should be recalled that there is uniformly associated with fibroid tumors a marked grade of hypertrophy and hyperplasia of the endometrium and that according to some pathologists, the squamous epithelium of the cervix exhibits similar signs of growth activity. Evidence is accumulating which shows an enormous concentration of the ovarian hormone folliculin in fibroid tumors, a fact which strongly supports the speculative assumption that such colossal cell proliferation as is exemplified in a rapidly growing fibroid tumor, and contemporaneously both in the hyperplasia of the endometrium and possibly also in the squamous cells covering the portio vaginalis, must be the direct result of sustained and powerful stimulation of a growth hormone. Whether or not such dysfunctional hormonal influence is responsible for an epithelial transition particularly favorable to the later development of cancer is but one of a number of related problems that await solution through the energetic and untiring efforts of our esteemed research colleagues.

The practice of coring out the mucous membrane of the cervical canal at the time of subtotal hysterectomy or destruction of it by heat applied in one form or another has been emphasized by some surgeons and adopted by a considerable number as a reliable safeguard against the subsequent development of stump cancer. But when it is recalled that more than 80 per cent of all cancers of the cervix originate from the squamous epithelium of the portio vaginalis it becomes evident that this procedure has only a meagre prophylactic value. On the other hand, the majority of stump cancers appearing within one year after subtotal hysterectomy are adenocarcinomas, and since these are assumed to coexist at the time of the operation, it becomes evident that the block of cored out cervical tissue possesses particular value for immediate biopsy by the frozen section technique.

Two other points of practical importance appear to have been established through statistical studies which need to be

emphasized because they are in conflict with prevailing surgical opinion: (1) that approximately 10 per cent of these stump cancers occur in women from twenty to thirty-five years of age; and (2) that over 20 per cent of these women have never been pregnant.

The latter point serves to focus our attention sharply upon the possible rôle which chronic infections of the cervix play in the etiology of cancer, since a considerable proportion of stump cancers follow subtotal hysterectomies performed because of the late consequences of uterine and adnexal infections. Furthermore, it has been abundantly demonstrated that such infected cervices are etiologically responsible for at least a small proportion of the cases of infectious arthritis. Chronic leucorrhea, which is so prevalent as to be accorded but scant consideration by the average doctor, is the sign that points unmistakably to the existence of these lurking menaces. Consequently, the teaching of those who emphatically condemn subtotal hysterectomy in the presence of chronic infection of the cervix is unquestionably sound.

Conspicuous in all of the discussions upon the universal adoption of routine total as distinguished from elective total hysterectomy has been the question of relative mortality. Unfortunately, on this phase of the subject statistics prove their own unreliability. Where they represent the operative results of highly trained, experienced and skilful surgeons the percentage difference in mortality between the two operations is so small as to represent only the accidents common to all branches of major surgery. In such hands the mortality in either group does not exceed 1.5 per cent. But according to the statistics of Fullerton and Faulkner based on 1851 consecutive hysterectomies, where the figures apply to the average run of an active clinic in which the routine operating over a period of years has been participated in by a score or more of individuals, including senior residents who have not yet completed their apprenticeships, the gross mortality in both types of hysterectomy is 4 to 4.5 per cent. Note, however,

that in 63 per cent of 1078 total hysterectomies performed by five members of the visiting staff the mortality was 3.5 per cent; while in 37 per cent performed by twenty members of the resident staff the mortality was 5.2 per cent. These figures indicate pretty clearly what would happen if inexperienced pelvic surgeons everywhere undertook the routine performance of total hysterectomy.

Furthermore, comparison of mortality statistics alone by no means tells the whole story. Certainly consideration of postoperative complications and of morbidity should not be omitted when one undertakes to evaluate the relative merits of two operative procedures. Notwithstanding this fact, there is a striking and disquieting paucity of data in the literature relating to this vitally important phase of the subject. But from my own observation and experience I have no hesitancy in asserting that if such specific tests as the incidence of operative and postoperative hemorrhage, surgical shock, damage to bladder, ureters and rectum with consequent fistula formation, postoperative crystitis, pelvic cellulitis, peritonitis, intestinal obstruction, wound infections, phlebitis, thrombosis and embolism, pneumonia, sustained daily elevation of temperature above 100° and total duration of convalescence were rigidly applied, it would be exceedingly difficult to justify the advocacy of universal routine total hysterectomy. Unquestionably this is a surgical procedure of far greater magnitude requiring larger experience and more highly developed technical skill for its successful application than does subtotal hysterectomy.

A discussion of vaginal hysterectomy does not come within the scope of this paper and I refer to it only in order to emphasize the fact that in properly selected cases it possesses distinct advantages over both total and subtotal hysterectomy performed by the abdominal route.

From what has been said it is apparent that the advocates of routine total hysterectomy offer three arguments in support of their contention; (1) the present incidence of stump cancer;

(2) the prevalence of focal infections in the cervix; and (3) the assertion that the difference in mortality between total and subtotal hysterectomy is so slight as to be a negligible factor.

Opposing conservative opinion, on the other hand, contends that the incidence of stump cancer should be interpreted merely as an index of the ill-advised use of subtotal hysterectomy, since one cannot discover in the recorded data any evidence that normal cervices later become cancerous; (2) that in the hands of the average surgeon total hysterectomy is unquestionably a more hazardous undertaking and is attended by a substantially higher mortality than is the subtotal operation, and (3) that likewise panhysterectomy involves far greater risk of serious operative and postoperative complications, as well as a longer period of morbidity, than does the supracervical technique.

Finally, no one can review the voluminous literature on this subject without being profoundly impressed by the continued reprehensible prevalence of benign diseases of the uterine cervix and their etiological relationship to cancer. Because of this fact it is exceptional to encounter a normal cervix in conjunction with the indications for hysterectomy. Consequently, despite its many advantages, conservative subtotal hysterectomy has today only a limited field of application. And recognition of this situation coupled with unsatisfactory experiences in connection with the older operations led me to develop a simplified technique for abdominal panhysterectomy designed specifically to guard against the major hazards incident to this procedure, namely, mortality, hemorrhage, shock, damage to ureters, bladder and rectum and postoperative peritonitis. Thus far in my own series, which as yet totals not quite 100 cases but which embraces every variety of both simple and complicated pathology requiring this type of surgical therapy, none of these hazards has materialized. Therefore, I can with great confidence heartily recommend this simplified technique to other surgeons who, like myself, have found the older operations formidable and unsatisfactory.

DISCUSSION ON PAPERS OF DRS. SHALLENBERGER AND RICHARDSON

Dr. F. Webb Griffith, Asheville, N. C.: Two years ago, before this Association, I reported the results of 693 hysterectomies done by me personally for benign conditions of the uterus. There were 14 deaths, a trifle over 2 per cent. In that series, all the deaths were after the subtotal operation. At first glance, it would appear from that that the subtotal is the more dangerous. I think it was Mark Twain who said that he considered the bed a most dangerous place, because so many of his friends died there. I feel that frequently our deductions are similar to those of Mark Twain. The explanation, of course, is simply that the poor risks were operated upon by the subtotal method. There is no doubt that in the hands of the average surgeon the supravaginal operation is by far the safer. If you take 1000 patients and do a subtotal hysterectomy on every one, and add to your mortality the deaths from cancer later developing in the cervix left behind, you will still have a much lower death rate than if all the thousand had been subjected to a total hysterectomy. In my series, most of which were subtotal, so far as I know only one patient subsequently developed a cancer of the cervix. Had there been any others, I think I would have known it from my work comes from a relatively small territory.

There is one other procedure that I think we might use in selected cases. For example, in a fat woman, where the pelvic organs are pretty well bound down, and where the cervix is lacerated and covered with a leucorrheal discharge, it is reasonably sure that a total hysterectomy would be quite difficult. In such a case, we are justified in doing a high amputation of the vaginal portion of the cervix, and then going into the abdomen and doing a subtotal hysterectomy, thoroughly cupping the remaining small segment of the cervix. That double procedure can be done with relative safety and in less time than the average total hysterectomy. In that way, all of the epithelium of the cervix, both columnar and squamous, has been removed and the danger of cancer eliminated.

Dr. Thomas S. Cullen, Baltimore, Md.: While Dr. Shallenberger was talking, I was reminded of an experience I had on a trip to Western Maryland. The hospital was close to the station and as soon as the train was heard approaching the anesthetist started the anesthetic and I had no opportunity to talk to the patient who entered the hospital to be operated on for a myoma which occupied the left side of the cervix and filled the greater part of the vagina.

On making a pelvic examination, in addition to the cervical myoma, I found an enlargement in the lower abdomen and at once said that the

patient was either pregnant or had an ovarian cyst. The family physician said that there had been no signs of pregnancy and that the patient had missed no period.

On opening the abdomen a four months' pregnancy was found. It might have been wiser to have closed the abdomen and waited until the pregnancy had advanced to term, but as the abdomen was open we went ahead, took out the cervical myoma and about one-third of the vaginal mucosa, brought the vaginal edges together and closed the broad ligament.

The patient went on to term and was delivered of a 10 pound child.*

On December 14, 1923, I saw a young woman who was five months pregnant and who had a ruptured appendix and a pelvic peritonitis. I at once removed the appendix, wiped out the pelvis, and placed two drains, one in the pelvis and one up toward the right renal pocket.

A few hours after operation the patient had labor pains. She miscarried the same night.

After operation her temperature remained normal but her pulse was a little rapid.

On December 23, her leucocyte count was 28,000 and she had a little vomiting, fecal in character. Her stomach was washed out and she was given an enema. A rather large loop of small bowel could be seen in the left lower abdomen.

I made a left gridiron incision and at once encountered free fluid. Three loops of small bowel directly behind the uterus had stuck fast to the promontory of the sacrum. We loosened these and the distended bowel at once grew smaller.

As a safeguard, a loop of small bowel was brought up into the incision to be opened later if required. This was, however, not necessary. The next day the patient's bowels were moved by enema.

The patient continued to have a fever, sometimes reaching as high as 103°F., and there was a definite thickening in the left side of the pelvis. This was opened on January 5, 1924, and about 50 c.c. of non-offensive, grayish yellow pus escaped. Two drains were introduced.

The patient made a slow but satisfactory recovery.

As she was very anxious to have children, I did a Rubin test from time to time and finally she became pregnant. She is now perfectly well and has two children.

I was much interested in the admirable and judicial manner in which Dr. Richardson handled the subject of subtotal versus complete hysterectomy for myomata.

In 1909 Dr. Howard A. Kelly and I in "Myomata of the Uterus" reported the results of a careful analysis of nearly 1700 cases operated

* The case is reported in full in Kelly and Cullen: Myomata of the Uterus, p. 532.

upon by us and by our colleagues. In this book we described in detail those cases where carcinoma of the cervix or body of the uterus was present at the time of operation, and also those cases where sarcoma was found developing in the myomata. We pointed out that in many cases the entire uterus could not be removed because of the dense inflammatory condition. After considering the subject from every angle, we came to the definite conclusion that taken all in all the supravaginal hysteromyomectomy yielded the best results, and I still feel that more patients can be saved by this method. Of course in those cases where at the time of operation the cervix is suspicious looking, then complete removal of the uterus is clearly indicated.

Dr. H. J. Boldt, White Plains, N. Y.: I propose principally to add verification to what Dr. Shallenberger has said with regard to pregnancy connected with myoma. Very many years ago a woman came to my office with labor pains. Upon examination I found that she was pregnant, between the third and fourth month, with an interstitial myoma as a complication. I did a myomectomy on her for the purpose of saving the child. She went to term with her baby.

With regard to Dr. Richardson's paper, I will say this: Whenever I found a pathologic condition of the cervix I did a hysterectomy; whenever I found a non-pathologic condition I did a supravaginal hysterectomy, because it was quicker, although I do not think I had any difference in mortality between subtotal and complete. I recall but one instance of a cancer developing after a stump was left in the uterus.

Dr. John S. McEwan, Orlando, Fla.: Since hearing Dr. Richardson's paper five years ago I have been following his technique in hysterectomy, and have been paying attention to the cervix. Our rule is to carefully inspect the cervix, and if it is apparently normal—no erosions, no ulcerations and no discharge—we do the subtotal hysterectomy. If it is not an emergency and the patient has a discharge we try to cure that before we do a complete hysterectomy. We have a large number of fibroids in the South among the colored people. I do not know how many we have operated upon, but a large number. When you get a fibroid in the negress with a couple of pus tubes, infections of the sigmoid and some other things, you do not do hysterectomy. I think the high mortality today in total hysterectomies is due to the operators. In traveling about this country and others I find a great difference in technique, and many of the complications come from men who are not careful in their work. They either operate too fast or do not know what they are doing. It is not necessary to have these complications. I have never seen a fistula following a total hysterectomy and believe that with ordinary care and a good technique one will not have them.

I think a complete hysterectomy is not indicated as a routine measure.

Dr. William T. Black, Memphis, Tenn.: I agree fully with what Dr. Richardson said regarding hysterectomies. In the Memphis General Hospital we run about four large fibroids a week, and we teach that the supravaginal hysterectomy is the operation of choice, for 90 per cent of these cases are complicated by inflammatory disease. In private work where conditions are different I more often perform complete hysterectomies. I think we should study the cervix before operating in an effort to determine whether or not the complete operation is indicated over the subtotal. Three lives out of every 100 will be saved, where less skillful surgeons operate, when the supravaginal hysterectomy is performed.

Discussing Dr. Shallenberger's paper: In the past two months have had two ovarian tumors complicating pregnancy. One was a twisted ovarian cyst the size of a grapefruit occuring in a patient six weeks pregnant. The corpus luteum of pregnancy was present in the ovary accompanying the cyst. In four or five days the patient aborted. Corpus luteum was given freely with the idea that the progestin might continue to act as it does during the menstrual cycle, by further keeping the decidua sensitized and helping in the continued nidation of the impregnation. Of course, one must consider that the progestin, which has this hormone effect, continues to be antagonized by oestrin, which is also developed from the corpus luteum. If progestin were available, many of these abortions could probably be prevented.

The second case was a dermoid cyst about the size of a six or seven months' pregnancy, which complicated a three months' pregnancy. The type of cyst was diagnosed preoperatively by *x*-ray. It is strange that the corpus luteum of pregnancy was also in the ovary in which the cyst was found. This patient did not abort, due to the fact that the placenta was able to carry on after removal of the corpus luteum of pregnancy.

These 2 cases are good examples demonstrating the stage in which the corpus luteum of pregnancy is essential, and the second case demonstrating that the function has been taken on by the placenta.

Dr. James F. Percy, Los Angeles, Calif.: I wish to speak concerning Dr. Richardson's paper. Just before coming to this meeting I operated, by my cautery technique, on a very extensive carcinoma of the cervix in a woman who had a subtotal hysterectomy twenty-three years before. It was for some non-malignant condition. I furnished 16 of the cases reported by von Graff, from whose paper Dr. Richardson has quoted, and in addition, found several more in my files. Postoperative carcinoma of the cervix following subtotal hysterectomy, therefore, is not an uncommon sequel in my experience, although I have no other record of this condition coming on twenty-three years later following partial removal of primarily

a non-malignant uterus. The women that I see with this condition rarely give a history of less than three years at the earliest before the characteristic symptoms of cervical carcinoma appear following the hysterectomy.

We all know that the vault of the vagina following a subtotal hysterectomy for fibroids is much more normal than it is following a panhysterectomy for cancer. The dangers of a subsequent carcinoma developing in a cervix after a subtotal hysterectomy can almost certainly be prevented if the cervical canal is thoroughly burned out from above with the actual cautery. This was advocated in a worthwhile article some fifteen or more years ago by a Southern surgeon whose name I have not been able to find for this discussion. It should then be emphasized that carcinoma of the cervix, following subtotal removal of the uterus, is much more common than we have heretofore realized, and we most earnestly advise the thorough cauterization of the cervical canal with the actual cautery (not diathermy) following the removal of the uterus.

Dr. W. F. Shallenberger (*Closing*): Some years ago I was called upon to testify in Court for the late Dr. Bert Wagnon, of Atlanta. He had operated upon a woman because of fibroid tumors. The patient had misled him in her history and was pregnant at the time of the operation but Dr. Wagnon did not suspect this and did not recognize the pregnancy until the time of the operation. He removed the fibroid tumors and the patient, after a fairly stormy convalescence, went to term and was delivered by another doctor. However, she brought suit against Dr. Wagnon.

When I was on the stand the attorney for the plaintiff said to me, "Is it not true that no operation should ever be done during pregnancy unless there is a good and sufficient reason?" I replied that "No operation should ever be done at any time unless there is a good and sufficient reason." He flared back with the statement "That isn't what I asked you. I mean should it not be a matter of life and death before any operation is done during pregnancy," and to this I replied that we should not wait until it is a matter of life and death before operating, and that is the attitude I wish to take regarding operations during pregnancy; namely, never operate unless there is a good and sufficient reason and do not wait until it is a matter of life and death.

INJURIES PECULIAR TO MODERN FOOTBALL*

LUCIAN H. LANDRY, M.D., F.A.C.S.

NEW ORLEANS, LA.

FLOYD R. Eastwood,[1] New York University instructor, who has conducted three annual surveys of football deaths and accidents, reported to the American Football Coaches' Association in Chicago on December 26, 1933 that over one-quarter of the injuries to college players during 1933 might have been avoided by "adequate leadership."

Although the survey, conducted for a coaches' committee headed by Dr. Marvin A. Stevens, former head coach at Yale, showed that the natural hazards of the game still were the major cause of all accidents, many of the most severe injuries and 27.8 per cent of the total could have been avoided by close attention to playing fields, coaching and the player's condition.

One hundred and seventeen colleges in thirty-eight states, with football squads aggregating 7084 players, cooperated in the survey for a good cross section of the college game.

The "preventable" injuries Eastwood listed under three headings: (1) those due to administrative control, such as inadequate coaching, poor playing field, 17.7 per cent; (2) training control, such as fatigue, carelessness, etc., 4.9 per cent; physical condition, 5.2 per cent.

From a study of these accidents, he drew the following conclusions:

1. Where the college administration pays for all injuries there probably will be a lower mean number of accidents per 1000 exposures.

2. Coaches who have had seven years or more experience have a lower mean number of accidents.

3. Coaches who have played the game themselves for three or more years will have fewer accidents.

* From the Surgical Department of Tulane University.

4. The coach who also handles some other sport will have fewer injuries than one who coaches only football.

5. Four weeks of pre-season training gives the lowest mean number of accidents and days lost.

6. Two days a week of fundamentals gives the lowest mean number of accidents and days lost.

7. A complete medical examination during pre-season training gives the fewest accidents.

8. Fewest accidents occurred where the coach or trainer determined the player's daily condition.

9. Colleges having a physician constantly in attendance showed the fewest accidents and days lost.

Of the 653 injuries which were possible of classification as to how received, Eastwood found that the largest number, 204, were incurred while the player was blocking or attempting to block another player, 154 were incurred in tackling, 119 by the player who was blocked, 72 by defensive linemen, 60 in line plunging and 44 by players tackled. Head concussions, the most dangerous of all injuries, were incurred only in blocking and while being blocked, and in defensive line play.

Only a small portion of the report was given over to fatalities, Eastwood reporting that only 28 deaths of 37 reported from all sources could be directly attributed to football injuries. Of these, 12 were received in sandlot play; 2 amateur; 12 high school, and 2 college. The college percentage, 7.2 per cent, showed a decrease from 16.3 per cent of the total in 1931, the first year the survey was made, and 9 per cent in 1932.

The survey for 1934,[2] published December 4, continued to show a decline in football fatalities from the high mark of 1931. The total deaths were 26, segregated as follows: college 1, high school 16, sandlot 6, club 3.

Dr. Thomas N. Horan[8] recently published a study of major and minor injuries through four consecutive seasons at Cranbrook School, where the attendance averages 200, 80 per cent of the boys taking part in football. His experience, as to type of

injury and region involved, closely coincides with our general average.

As for longevity of college athletes,[3] Dublin in *Harper's Monthly Magazine*, July 1, 1928, gave the following interesting statistics in relation to athletes, nearly 5000 in number, who lived a total of 146,426 years from graduation in 1905 or before; and athletics at ten leading American colleges. These statistics show that the mortality rate among baseball players is greater than that of any other sport, with crew next, followed by track. Football has a mortality rate which is better than the average expectancy of a non-athlete. Men playing two or more sports seem to have the best expectancy. Further studies by Dublin showed that Phi Beta Kappa men lived longer than athletes and that Harvard men live longer than Yale men, but the mortality rate is high among Harvard athletes and very favorable to Yale athletes. One-fifth of all deaths in younger men were caused by accidents after escaping fatalities throughout a strenuous sports career.

Stevens and Phelps[3] in the preface to their book state: "It might be well to point out that in our experience it is definitely more dangerous to drive to the Yale Bowl as a spectator than it is to play football the entire season as our accident rooms are frequented much more often by spectators than players."

College football, like the automobile, has made tremendous strides in the way of "improvements" during the past twenty-five years, and has likewise increased in popularity both with the players and the audience. Like the automobile, the steady and progressive trend of the game has been for speed, speed and more speed. This largely explains the necessity of the great numbers of players the representative colleges put on the field. It is not unusual to see from thirty-five to fifty men on the squad of the smaller universities and as many as 100 or more on the squad of the larger universities. (The West Point squad is restricted to 84 by regulations; the Yale freshman squad averages 100 men.)[3]

A most notable exception to this dictum was registered last month, when Yale played and defeated Princeton without making a single substitution, a very fine showing for training and condition. This was so striking and noteworthy that it was commented upon and lauded by practically all the major newspapers of the country, who referred to Yale as the team of "eleven iron men."

In the vast majority of instances, as soon as a man is noticed to be slowing up either from fatigue or a possible minor injury, he is immediately withdrawn from the game, his place being taken by a fresh, strong, man, to keep the team or machine geared up to high speed. A tired or slightly injured or loafing player in more liable to injury than a fresh man, as an alert player absorbs the blows with tense muscles, thereby minimizing the possibility of injury.

The gradual though constant cry for speed has not only modified the game from the old type of play when a team was given three downs or trials to make five yards (1906) to the newer form (1912) where four trials are allowed to gain ten yards.

The so-called "open play" brought about by the use of the forward and lateral passes has been a great factor in developing speed in the game and has also served to change the type of player used nowadays. We don't see the "overstuffed sofa" of 250 and 300 pounds playing center or guard; he is too big and slow for the modern game and has been superseded by the "streamlined" 150 to 190 pound player who is alert and fast enough to get out of his own way.

The necessity of speed has altered the entire equipment of the player from the heavy bulky and cumbersome outfit (including quilted pants) that weighed anywhere from 15 to 20 pounds and much more when playing in the rain, to silk pants with no padding and very light shoes, no stockings and thin jerseys, the entire outfit rarely weighing more than 10 pounds. The heaviest piece of equipment that a player wears today is the shoulder pads, a piece of protective mechanism for

the clavicles, shoulders (?) and upper chest made of well-moulded leather lines with sponge rubber. (We feel that further improvements can be made in this piece of equipment.)

These changes in style of play and type of equipment has led to a different variety of injuries. We see a greater number of joint injuries, chest injuries (lower chest) and hematomas, especially of the thighs, legs, arms, shoulders and forearms.

The two most hazardous maneuvers in the technique of the game are: (1) blocking and (2) tackling. The so-called flying block and flying tackle (which was made illegal by the Rules Committee in 1932) was still more dangerous to the blocker or tackler, especially if he failed to hit the mid-thigh of the moving target. If he dove too low, he was very apt to put his torso or head in the line of a pair of hard driving knees, or if still lower, to one of two fast moving feet. If the diving tackle was made from behind and the tackler missed his mark, he was apt to receive a fractured nose from a heel, or an acromio-clavicular dislocation by landing on the point of the shoulder.

JOINT INJURIES

Football would have few injuries indeed, if all the joints could be adequately protected. Joint injuries are associated with ligamentous tears to a greater or less degree.[3] "Ligaments are the limiting structures around joints. They are thickenings in the joint capsule which prevent abnormal mobility taking place. When the force applied to a joint is sufficiently great to bring about abnormal mobility, a tear of the ligament results." There is always some bleeding associated with the tear which may be intra-articular or extra-articular or both. All joint injuries sufficient to cause ligamentous tears should be *x*-rayed for fear of missing an associated fracture running into the joint.

The joints most susceptible to injury in order of their frequency are: knees, ankles, shoulders.

Knees: The most vulnerable spot of the player cannot be adequately protected by padding or a brace without materially

interfering with free motion and thereby checking his speed. The knees are most often injured by the player being blocked or tackled from the side, especially if the knee is struck by the body or shoulder of the tackler just as the entire weight of the body is applied to that particular limb; the foot being practically fixed to the ground, the leg, instead of being knocked from under the player, is bashed in. This frequently results in a stretching or a tear in the internal lateral ligament, and occasionally the semilunar cartilage as well. The anterior crucial ligament may be ruptured by this manuver if the force is directed from the front. This, however, is a rare injury unless the trauma is severe enough to cause complete dislocation. Ordinarily, the semilunar cartilage is torn, dislocated or split by a twisting or turning motion with the foot fixed and is more common in baseball, caused by overswinging at bat or in rounding the bases at a high rate of speed. The internal semilunar cartilage is affected far more frequently than the external; the ratio is 15 to 1 (Campbell[4]). We have had 9 cases of semilunar cartilage tears; 6 were operated on and returned to the game the following year.

The internal lateral ligament tears are followed by severe pain, marked swelling, effusion, ecchymosis and inability to flex or extend the leg. Pressure on the outside of the knee with the muscles relaxed increases the pain. These injuries are best treated by cold packs if seen immediately, followed by elastic compression bandages, hot packs, crutches and possibly aspiration; diathermy is used later; in severe cases a light plaster cast may be necessary. Early walking is encouraged, avoiding deep knee flexion.

It is surprising at times how quickly some of these knee injuries will subside by the liberal and frequent use of hot packs and compression bandages and the boy be allowed to return to the game wearing a specially constructed ball bearing steel or aluminum brace with a leather lacer above and below the joint, which is adequate protection against recurrence of the injury.

Ankles: The ankle is subject to constant danger as it may be twisted or turned in running without the necessity of being struck by another player. This injury is more apt to occur on a playing field that is not properly cared for; we find more turned ankles while the practice field is being used, as the field is constantly being cut up by the steady and repeated abuse of the multicleated shoes of a large squad. It rarely occurs on the field that is used only for weekly contests.

I firmly believe that a properly strapped ankle cannot be seriously injured unless there is sufficient force exerted to cause a fracture. It is not practical to strap every boy's ankles every day before he goes to the practice field; this, however, is done on the day of the game. Many of the players use "ankle wraps" for scrimmage; these are simple light canvas bandages bound about the ankles and are, to my mind, no more than psychologic props.

As you must know, it is quite warm for the first half of our football season; in the past we had considerable difficulty making our adhesive straps stick through the first half of the game; of course, the lower third of the leg was shaved, but the inevitable perspiration invariably loosened the straps. In the past three years we have had no trouble from this score, thanks to the ingenuity of Mr. Claude Simons (trainer) who conceived the idea of painting the lower third of the legs with tincture of benzoin, which is not only a good antiseptic, but allows the adhesive to get a firm grip on the skin; a snug light roller bandage is applied over the adhesive tape to insure close coaptation. This has been very satisfactory in our hands and I wish to recommend it highly.

Unfortunately, in many institutions, the prophylactic strapping of joints is left entirely in the hands of the trainer or coaches; if they have not been properly instructed in the art, they can do considerably more harm than good. I have seen ordinary electric friction tape used as a figure of eight around the ankle, which undoubtedly interferes with foot flexion and extension, as well as impedes the venous circulation. A properly

applied modified Gibney splint allows unrestricted flexion and extension, but prohibits almost entirely any lateral motion in the ankle. If lateral motion is prohibited, a tear or severe stretch of the lateral ligaments is impossible.

Acromioclavicular Dislocation (Football Shoulder): The distal end of the clavicle is forced upward away from the acromion process of the scapula resulting in a rupture of the acromioclavicular ligament and possibly the coracoclavicular ligament as well, which normally hold the clavicle down. This condition is often referred to as a "knocked down shoulder" by coaches and trainers; it should be called a "knocked up shoulder" as the end of the clavicle at times rides as high as an inch and a half higher than its normal position. The injury may occur as a result of a fall sideways on the elbow or wrist with the arm outstretched, the same maneuver that produces a shoulder dislocation. More often it is caused by a missed tackle, the point of the shoulder striking the unresisting ground and forcibly shoving the distal end of the clavicle backward.

This is the most frequent injury about the shoulder, the arm being put out of commission, as all movements of the arm result in pain. Pain is also experienced by pressure on the elevated distal end of the clavicle for days and sometimes weeks following the injury. The arm is immobilized for three to eight days with frequent treatments of moist and radiant heat, after which gradually increasing use and exercise are allowed. The outer end of the clavicle is then protected by the use of rubber sponges and adhesive straps. When the arm can be used without pain, a special leather moulded brace (horse collar) is fitted over the entire shoulder that prevents pressure on any portion of the clavicle.

I know of only one boy who complained of inconvenience in the shoulder after the cessation of his football activities. I have never used the Watkins or other surgical procedures to correct this deformity. Dr. E. D. Fenner,[5] of New Orleans, has had 4 cases operated on with good functional results; however, I know of no boy operated on who returned to the game.

Dr. T. E. Christian[6] of San Antonio, Texas, recently gave a moving picture demonstration at the Southern Medical Meeting, covering the operative and nonoperative treatment of this lesion, stressing the point that a great many of these patients will recover without operation.

The sternoclavicular dislocation is comparatively rare in football; it is due to a direct blow or extreme backward twisting of the arm during a fall, pulling the sternal end of the clavicle out in front. I have seen but 2 such cases.

Dislocations of the shoulder or elbow are rare; when either of these conditions is found, it is usually ascribed to a clumsy or incorrectly executed maneuver.

HEMATOMAS

Hematomas are rather common, owing to the very scant padding worn by the players. The most frequent site is the anterior and antero-lateral aspect of the thighs; the legs, arms, forearms and shoulders follow in order of frequency.

These must be differentiated from a "Charlie horse," which is essentially ruptured muscle fibers, generally associated with a certain amount of hematoma.

The treatment of both conditions is practically the same: heat, rest, compression bandage and *no massage*. Hematomas near the crest of the tibia are apt to become infected, due to tissue maceration, and may need incision and drainage.

MYOSITIS OSSIFICANS

Campbell[7] reports 7 cases following football injuries (5 left thigh, 1 right thigh, 1 left arm), 3 cases treated surgically; however Campbell does not advocate open operation; he rather recommends rest, moist heat, early motion (walking) but no massage.* I have had no personal cases in my experience.

Brush burns, bruises and cuts are treated immediately; very little tetanus or perfringens antitoxin has been used in

*Campbell has had 2 additional cases, non-operative, since the publication of his paper.

our group; I have never seen a case of tetanus or gas bacillus infection follow a football injury.

FRACTURES

Unfortunately, we still have too many broken noses (four this year). Attempt at reduction is made immediately and the case put in charge of the rhinologist who finds that better results are obtained when the reduction can be effected before a great deal of edema and swelling sets in. The vast majority of these boys are allowed to play the following week, the nose being well protected by a very strong specially constructed nose guard which is firmly fixed to the head. This nose guard is far superior to the old hard rubber type that was very insecurely held by an elastic band around the head and also by the front teeth of the player. I know of no way to prevent fractures of the nose, as the players will not wear the nose protector unless they have sustained a fracture.

Fractures of the superior maxilla (one this year) and zygoma are the result of a direct blow from a head, knee, foot or elbow; these players are usually returned to the game in two or three weeks, but are thoroughly protected by a complete heavy mask built into the head-gear, making it impossible to strike or hurt the injured part again.

Note: While the head gear is adequate protection against scalp, ear and cranial injuries, it is too hard and becomes an instrument of danger, causing more injury to the opponent than protection to the wearer.

Fractured fingers (2 this year) can be thoroughly protected and immobilized in such a manner that a player may be returned to the game in one or two weeks with a minimal risk of reinjuring the member. One player this year received a longitudinal fracture of the proximal phalanx of the right little finger; with the aid of a moulded metal splint, he was able to throw and receive passes in a game one week after his accident. Another player with an impacted fracture of base of the first metacarpal of the right hand, played two weeks after

his injury, the thumb being firmly fixed in a metal splint and steadied by the index finger; he was able to catch a forward pass. Another player (a tackle) with a fracture of the radius, mid third, no displacement, who was most anxious to play in a game two weeks after receiving his injury, was allowed to play, wearing a perforated leather steel reinforced brace of the forearm and hand; he made a very creditable showing and did not reinjure the forearm.

These illustrations are given, not as an example of rare stoicism on the part of the player, or of super-efficiency on the part of the team surgeon, but more to bring out what can be accomplished in the way of fixation and protection of injured parts by the use of modern, light and rigid splints that are not as cumbersome and bulky as their predecessors, the board and plaster splint.

VISCERAL INJURIES

Visceral injuries are rare enough to be classed as accidental injuries; while I have had 8 cases of hematuria following a blow over the kidney (6 in college games) which were classed as kidney lacerations, only 2 required nephrectomy. One boy (from another school) returned to the game the following year against instructions; he fortunately received no further injuries.

CONCLUSIONS

Finally, I wish to make a plea to the young surgeons to pay more attention to these ever-increasing football injuries, as more and more boys are taking part in the game. These injuries should not be left to the care of trainers and coaches. While I will admit that some of the experienced trainers in the larger universities have become good diagnosticians and can give good first aid treatment in most instances, the sooner the injured boy is seen by a surgeon, the less likelihood there will be of a disabling injury.

High schools and preparatory schools are taught football by young coaches; they may or may not have a trainer, but very few have a regularly appointed surgeon to care for their injured. A large field is opening for the young man in surgery, as football is not only here to stay, but is on the increase.

TABLE 1

INJURY EXPERIENCE AT TULANE SEASON 1934

Ligamentous:	
Knees	10
Ankles	8
Shoulders	7
Hip	1
Bone (Fractures):	
Nose	4
Sup. maxilla	1
Fingers	2
Tibia	1
Radius	1
Scapula	1
Muscles:	
(Hematoma)	6
Skin:	
(Lacerated or incised wound needing suture)	5
Ruptured ear drum	1

Note: For a full statistical study of fatalities and injuries, including nature of injury and manner in which injury was received see tabulations in Stevens and Phelps.[3]

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DR. GEORGE E. BENNETT (Baltimore, Md.): I think lay people have the idea that football is a dangerous game. As a matter of fact, it is not dangerous. Many of the points mentioned in Dr. Landry's paper are imperative, particularly the protection of the joints preparatory to football practice. Fortunately, in many of the universities there are doctors who see that the ankles are properly strapped, and I think if they would see to it that the boys are properly protected during practice as well as in the game it would be a great advantage.

SUPPURATIVE PERICARDITIS

GEORGE H. BUNCH, M.D., F.A.C.S.

COLUMBIA, S. C.

ALTHOUGH suppurative pericarditis may have been found in cadavers even by the early anatomists its acceptance as a practical clinical entity has taken place within the last twenty-five years. The notable contributions of Matas,[1] Rhodes,[30] Winslow and Shipley,[2] to the subject reflect credit upon the Southern Surgical Association. Shipley has the distinction of having operated in 13 cases, with 7 recoveries, a greater number of cases than has been handled by any other surgeon. In 1927 he, with Winslow, made a comprehensive study of the subject. From 118 cases found in the literature and 10 new ones added by them they gave to the profession a new understanding of the prevalence of the condition, its symptoms, its gravity and its treatment. Without reference to historical detail, in this paper, we shall endeavor to review briefly the present knowledge of the disease and to report 4 personal cases.

The incidence of the disease is undoubtedly much greater than the less than 200 operative cases reported in the literature up to this time indicate. Stone,[3] in 1919, reported finding in 300 autopsies done on soldiers dying of pneumonia at Fort Riley 24 per cent with pericarditis and 15 per cent with pyopericardium. In Stone's series the incidence of pericardial involvement was greater than usual for most of the patients must have been victims of the influenza epidemic then raging and cannot be considered as being ordinary cases of pneumonia. This is borne out by the report of Brooks,[4] in October 1934, who, in autopsies done on 200 consecutive individuals dying of pneumonia in Bellevue Hospital, found 75 with positive blood cultures or pathological signs of septicemia and not one with purulent pericarditis. Its ordinary incidence in individuals

dying of pneumonia must be somewhere between these two extremes. Musser and Norris,[29] in 1907, in a collected series of 2128 autopsies of patients dying of pneumonia found acute pericarditis in 12.6 per cent. If we accept Scott's estimate that 40 per cent of these cases are suppurative the incidence of pyopericardium is about 4 per cent.

Pyrah and Pain[8] in 796 autopsies done at the Leeds General Infirmary, 1921–1931, found 214 cases of acute pericarditis of which 91 were suppurative, 11.5 per cent. Pneumonia was given as the primary focus in only 8 cases although 45 per cent followed intrathoracic disease.

Non-traumatic suppurative pericarditis is essentially secondary and most frequently follows respiratory disease, particularly pneumonia. Septicemia, rheumatic fever and osteomyelitis may also be complicated by it. In 62 cases in which bacteriological findings are reported, by Winslow and Shipley, various types of pneumococci were identified in 28, of streptococci in 13 and of staphylococci in 13. Twelve followed gunshot or stab wounds of the chest.

Although the pericardium is described as being a fibrous unyielding sac it is remarkable how it may be distended by the gradually increasing pressure of an effusion. Winslow and Shipley[2] say that the amount of pus may vary from a few drops to 7500 c.c. Cutler[12] has aptly stated that there is a delicate pressure relationship between the pericardium and the right auricle. "The pressure in the right auricle during diastole is very slight and the pressure in the venae cavae on inspiration may be negative." As pericardial effusion increases either the pericardium must yield and relieve pressure on the auricle or the flow of blood to the heart will cease. When the intra-pericardial pressure approaches that of the blood in the right auricle acute circulatory failure from cardiac tamponade results. This is a usual complication of stab wounds of the heart where there is sudden hemorrhage into the pericardium and no time for pressure accommodation. In pyopericardium mechanical pressure on the heart depends more upon

the rapidity with which the effusion forms than upon its volume.

The symptoms of pyopericardium are those of sepsis plus those of impaired circulation from mechanical embarrassment to heart action due to increasing pressure made upon the heart by the accumulating effusion. When infection of the pericardium occurs there is apt to be precordial pain with exacerbation of septic symptoms already present. There is more often, however, continuation of symptoms in a patient who is obviously not doing well. There may be chills with high fever. There is always dyspnea which may be extreme.

The diagnosis of pyopericardium, although primarily the responsibility of the clinician, remains a challenge to the profession, for effective treatment depends upon early recognition. Osler said, "No serious disease is so frequently overlooked." According to Truesdale[5] (1933) at the Boston City Hospital only 17 per cent of 153 autopsy cases were correctly diagnosed. In Poynton's[6] (1908) series of 100 autopsy cases in children only 6 were diagnosed. One should always have the condition in mind when a case of pneumonia or septicemia does not do well and frequent examination of the chest should be made. Diagnosis should not be more difficult than in encapulated empyema. Symptoms are only suggestive but physical signs are characteristic.

Physical examination is typically that of an acutely ill patient, toxic and weak from pneumonia. There is shortness of breath and cyanosis in varying degree from venous congestion. Two of my 4 cases had general edema. The pulse is rapid. The lung findings depend upon the stage of resolution at the time of pericardial involvement and upon the degree of cardiac decompensation. The heart sounds are usually indistinct as if muffled and the apex beat is weak and hard to distinguish. In my first case, however, the sounds were accentuated as if the heart were floating in the effusion and in immediate contact with the chest wall. There is precordial dullness varying in extent upon the size of the effusion.

Posteriorly there is dullness over the mediastinum and to the left. At some time a definite friction rub may be distinguished in most cases.

Electrocardiographic tracings[27] are not of diagnostic value. In spite of the nominal danger of puncturing the heart or of injuring the coronary vessels with the needle careful paracentesis is indicated in effusion to determine the character of the fluid. Roentgen examination of the chest should always be made. When there is an enlarged area of precordial dullness shifting with change of position and when *x*-ray examination show a water bottle shadow with the base below there is present a pericardial effusion.

The mortality of purulent pericarditis treated medically is practically 100 per cent. In 152 operative cases found in the literature by Truesdale[5] in 1933 the mortality was 42 per cent, a reduction from 65 per cent in the first 52 operative cases. The disease is largely limited to childhood and to early adult life. Of Truesdale's 127 cases in which the age is given 11 were under five years of age with a mortality of 45.5 per cent, 29 were between five and ten years of age with 35 per cent mortality, 39 were between ten and twenty years with 40+ per cent mortality, 26 were between twenty and thirty with 38+ per cent mortality, 18 were between thirty and forty with 50 per cent mortality and 5 were over forty with 40 per cent mortality. The oldest patient was fifty-seven, the youngest one year. The age of the patient seemingly has but little effect on the mortality rate. According to Winslow and Shipley[2] there has been 30.8 per cent mortality in cases infected with streptococci, 37.03 per cent in cases infected with pneumococci and 54.8 per cent mortality in the staphylococcic group. The strength and the resistance of the patient at the time of the development of the complication has a direct bearing on the mortality.

Although suppurative pericarditis most often follows pneumonia it must not be forgotten that there may be other complications of the disease. The most notable is empyema,

which requires special treatment and which increases the mortality. Winslow and Shipley[2] state that in the 128 cases reviewed by them empyema was diagnosed and drained in 16

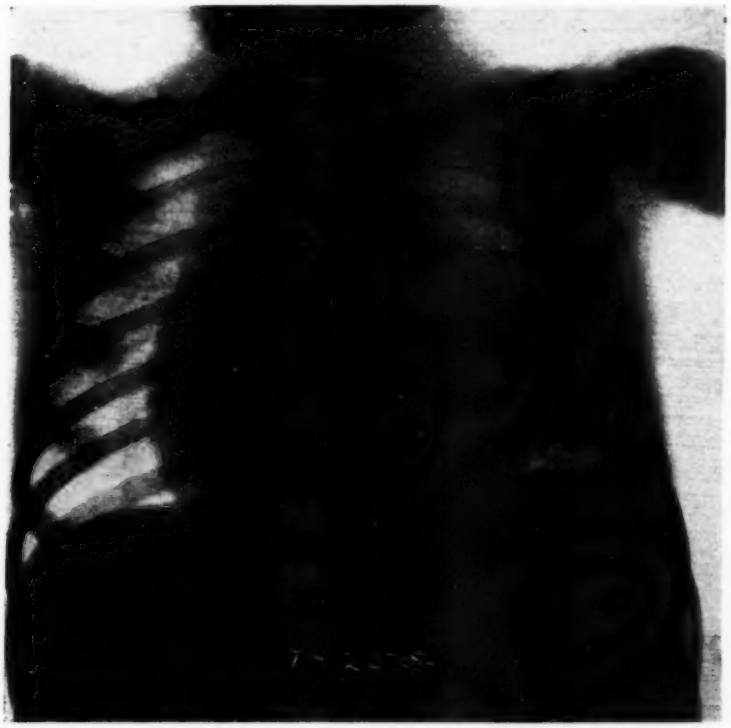

FIG. 1. Case I. Water bottle shadow of pericardial effusion with encapulated left empyema.

patients, of whom 7 recovered and 9 died following thoracotomy. In 8 cases the condition was found only at autopsy, so that of 24 patients known to have had empyema as a complication 17 died. In 62 of 72 autopsies on individuals dying of pneumonia complicated by purulent pericarditis empyema was found by Stone[3] as an additional complication. All of the 4 operated upon by me had empyema. In Case I it was coexistant with pyopericardium and in Case IV developed after pericardiotomy; both patients recovered. In Case II it existed before pericardiotomy and the patient died. In Case III the patient had an encapulated empyema drained by thoracotomy before pericardiotomy and a second encapulated empyema on the left side posterior

to the pericardium subsequent to pericardiotomy. The second was diagnosed only when the patient was almost moribund. It was shown by *x*-ray, proved by paracentesis and demon-

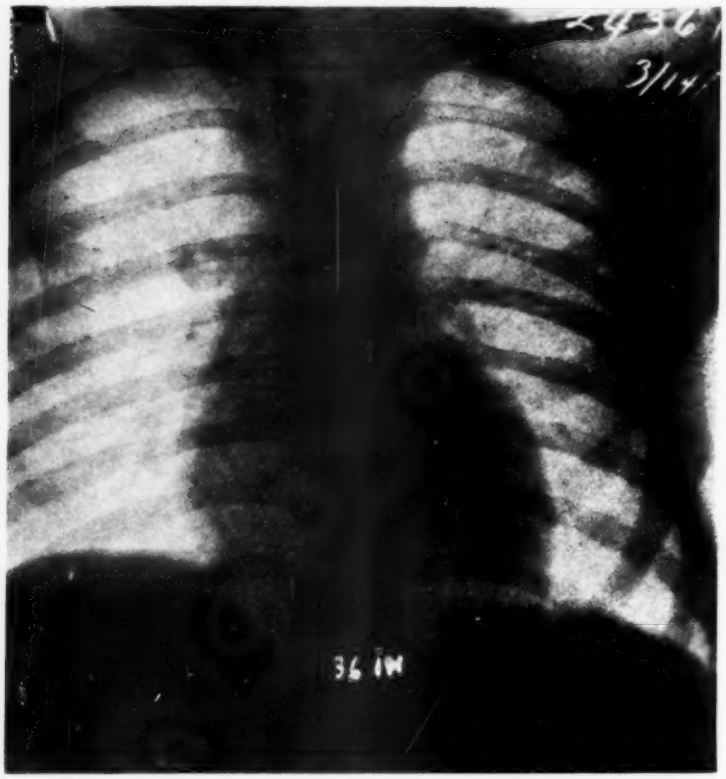

FIG. 2. Case I. Four and one-half years after operation. Normal heart.

strated at autopsy. If it had been diagnosed earlier and if his condition had been better the empyema might have been drained through an anterior approach. Blood culture in Case IV was reported positive for staphylococci.

The treatment of suppurative pericarditis is adequate dependent drainage by open operation as soon as the diagnosis is made. This is best done under local anesthesia. It is easily accomplished and it is safe. There is so little shock that it is but little more than a minor procedure. The approach to the pericardium should be through the interpleural space, the so-called triangle of safety. This is behind the sternum and is

pictured[12] as extending irregularly to the left. The apex is above at about the level of the fourth costal cartilage and the base is below at the level of the base of the xiphoid. The space

FIG. 3. Case II. Before therapeutic paracentesis.

varies with the size of the heart and is somewhat larger in pericardial effusion. In my cases I have made a hockey-stick incision along the left sternal margin and by resecting the sixth costal cartilage have exposed the pericardium which is grayish in color and thicker than the normal pleura. At this level if the incision into the pericardium is made near the sternal margin it will usually miss the left pleura and will be median to the internal mammary vessels which may, if necessary, be retracted laterally or ligated and divided. We have drained every case with a wick of folded rubber dam extending into the pericardium and secured with a safety pin through the skin. The soft wick keeps the drainage tract open without irritating or injuring the heart. We have not irrigated the wound. Pulsation of the heart insures drainage of the pericardium if no encapulated

pus pockets are present. This is facilitated by posture if the patient is kept on a back rest. No patient not moribund is too sick for operation. The hope of cure is in early drainage.

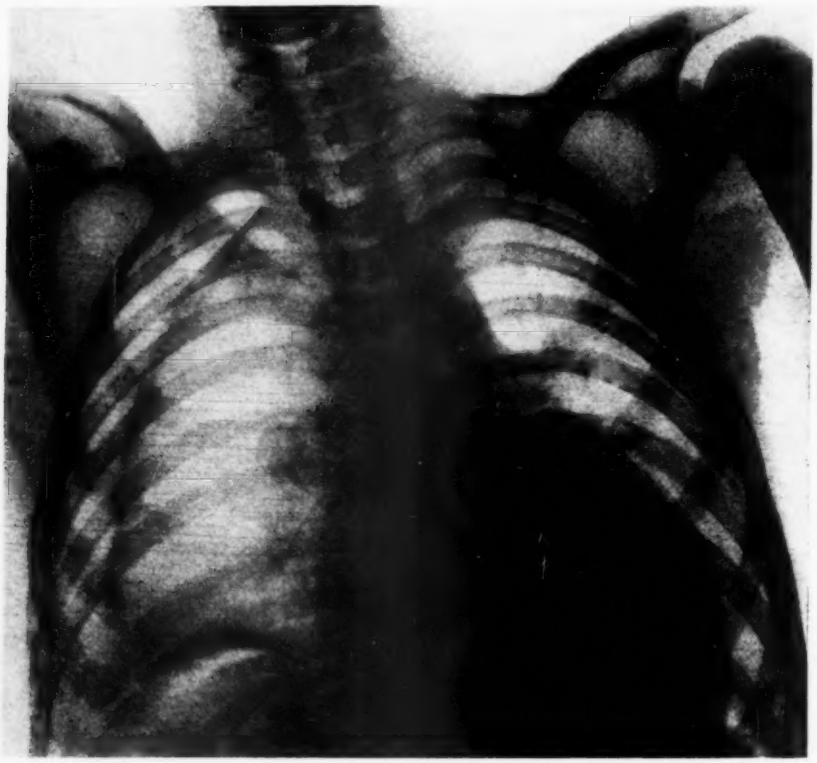

FIG. 4. Case 11. After repeated therapeutic paracentesis. Before operation.

Paracentesis is essentially a diagnostic and not a therapeutic procedure. After pericardiotomy, if septic symptoms continue or return after improvement has begun, an encapulated pus pocket should be sought for in the posterior pericardium or in the pleura. Left undrained such a pocket will surely kill the patient. We have gone a long way since Stone,[3] in 1919, reported having done autopsies on 72 cases of pyopericardium, none of which was operated upon. He significantly said, "The more radical procedure of incision and drainage of the pericardium in purulent pericarditis was not considered advisable in any of these patients in which the condition was sus-

pected because of the extreme pathological condition." And again, "If one might be so fortunate as to recognize purulent pericarditis coexistent with or subsequent to empyema and the

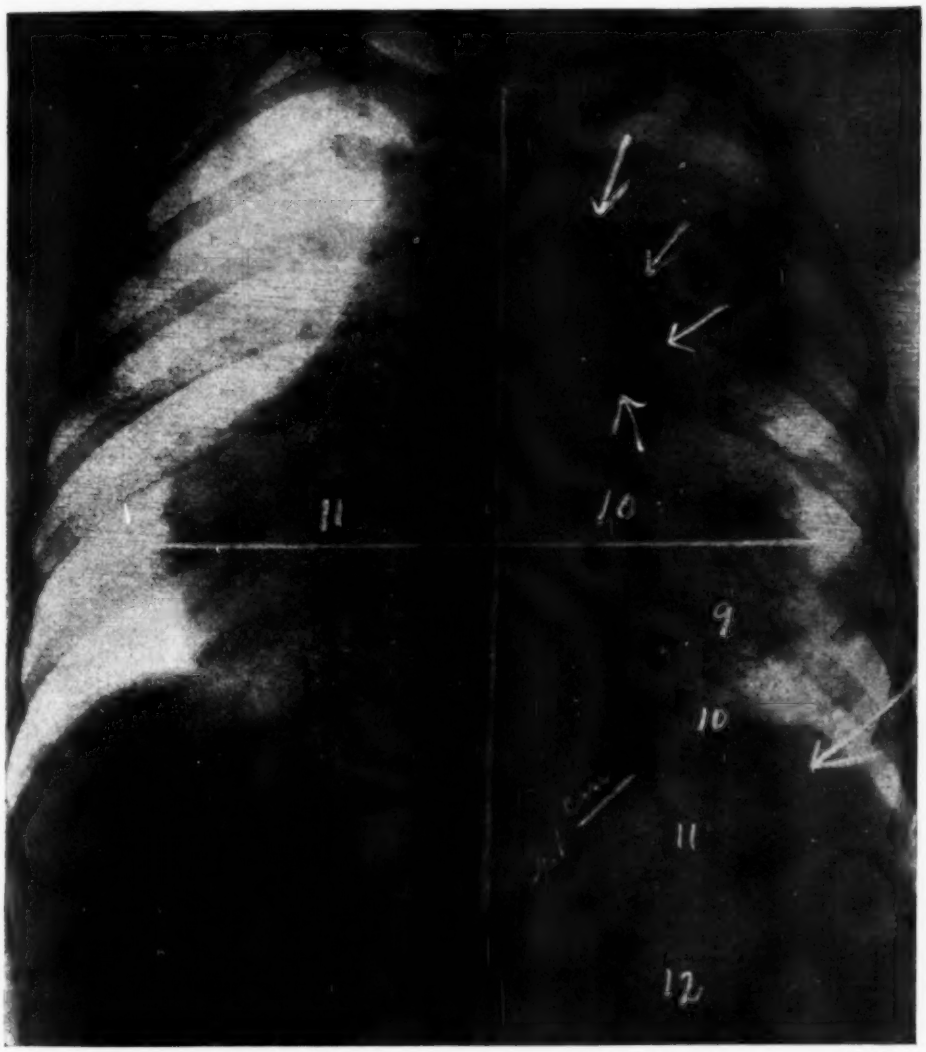

FIG. 5. Case III. Pericardial effusion. Left empyema drained by thoracotomy, below. Left encapulated empyema above.

condition of the patient was such as to warrant further interference, free incision of the pericardium would be indicated."

After recovery from pericardiotomy there are no data available as to the incidence of pericardial adhesions. Certainly

many cases have normal heart function. This, in my 2 cases, has been confirmed by physical and *x*-ray examination and by electrocardiographic study.

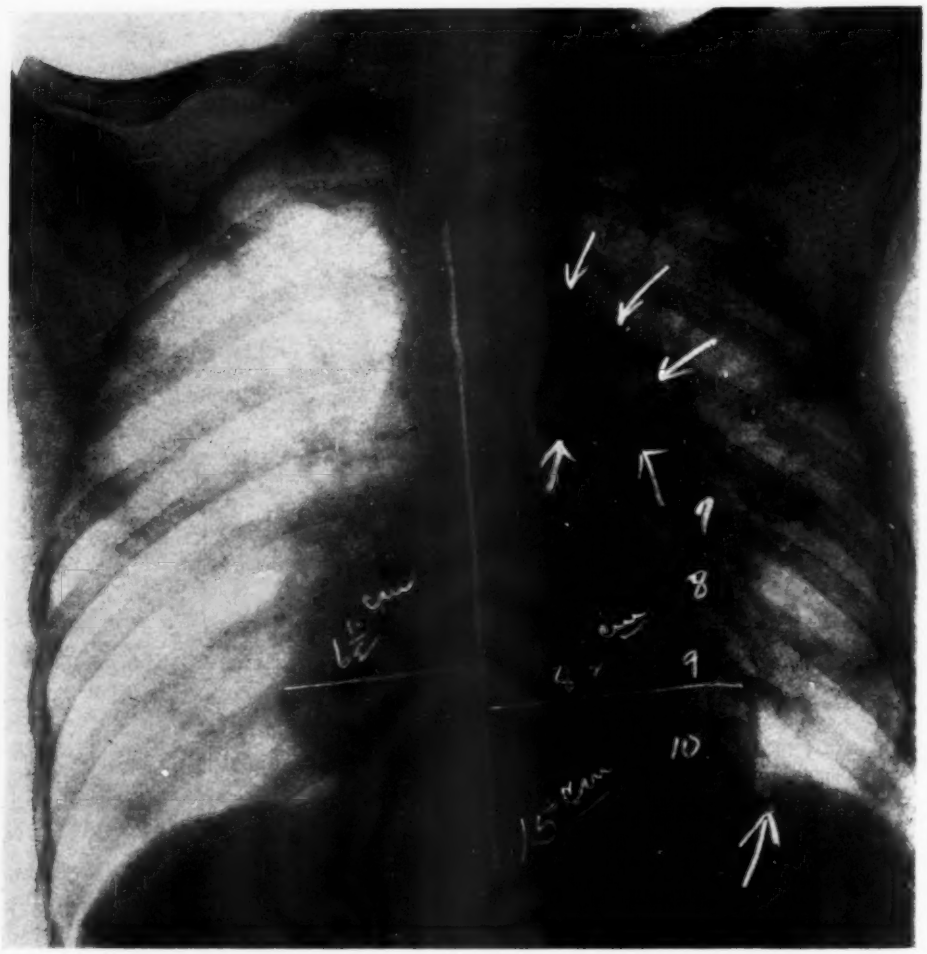

FIG. 6. Case III. Three weeks after pericardiotomy. Heart shadow, much smaller. Encapulated empyema, upper left side, undrained, caused death.

CASE 1. Baby J. H., white boy, aged nineteen months, was admitted to the South Carolina Baptist Hospital September 28, 1929 with leucocytes 18,000, polymorphonuclears 53 per cent, hemoglobin 35 per cent, rectal temperature 97.2°, pulse 130, respiration 40. He was normal at birth but has not been well since an attack of whooping cough when about six months old. He has been acutely ill since an attack of pneumonia beginning early

in September. There has been progressive loss of weight with dyspnea and weakness.

Examination shows a pale emaciated child with edema of the face and

FIG. 7. Case IV. Suppurative pericarditis effusion.

legs. There are crepitant râles over the left chest with dullness in the left axilla. Precordial dullness is enlarged. No friction rubs are heard. The heart sounds appear accentuated and the apex beat is in the normal position. X-ray of the chest shows a large shadow in the cardiac area of water bottle shape and also a shadow on the left indicating an encapulated empyema. Paracentesis pericardii was done and pus obtained. On September 29 the sixth costal cartilage was resected under local anesthesia and several ounces of thick white pus escaped when the pericardium was opened. The wound was drained with a small piece of folded rubber dam. On October 7 the encapulated empyema on the left side was drained by thoracotomy.

Pneumococci were found in the pus from both.
The child did nicely and was dismissed without fever October 28, 1929.
Physical examination, March, 1934, shows a healthy boy of good color

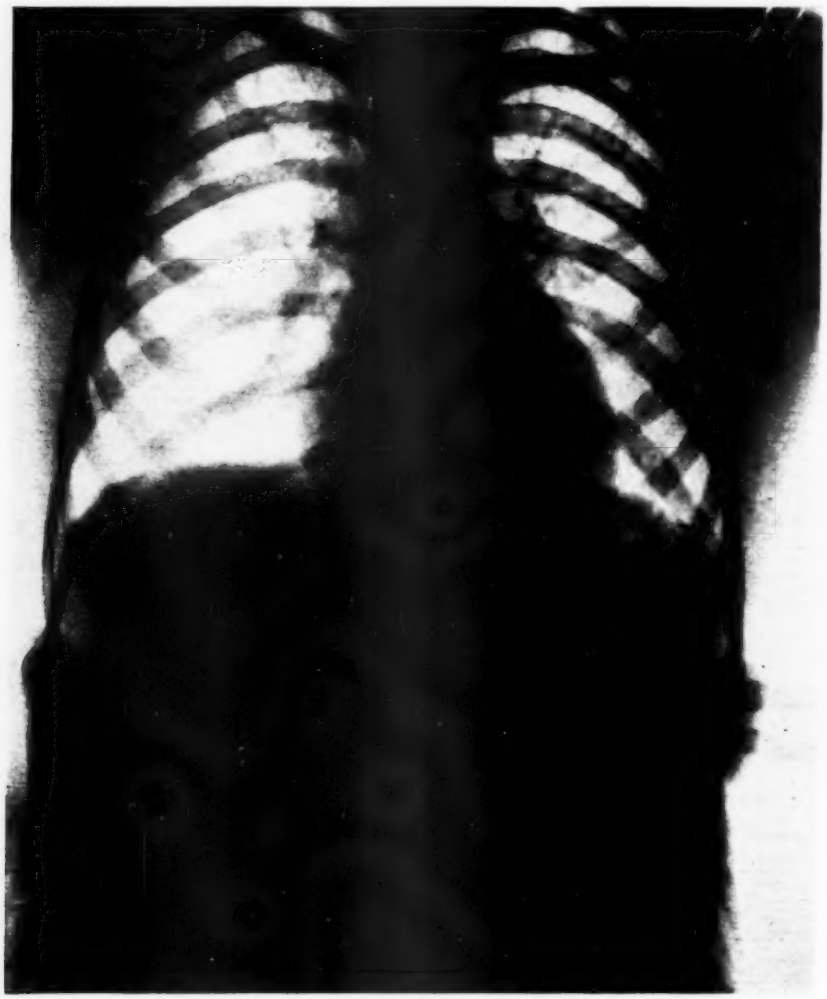

FIG. 8. Case IV. Normal heart shadow, one year after operation.

with normal heart action, normal electrocardiographic tracing and a normal heart shadow.

CASE II. D. J. S., white boy, aged eleven, was admitted to the Baptist Hospital March 18, 1931 with an encapulated empyema in the right base following influenza and lobar pneumonia. His temperature was 101.6°F., respiration 54, pulse 134, urine normal, leucocytes 17,800, with 74 per cent polynuclears. He was dismissed April 4 and did well at home

until April 23 when respiratory embarrassment developed with swelling of the legs and general prostration. On readmission to the hospital April 26 his pulse was 130, respiration 22, temperature 101°F., leucocytes 26,000,

FIG. 9. Case of congenital heart. Acute bronchitis, fever and leucocytosis, showing difficulty of diagnosis without paracentesis.

polymorphonuclears 78 per cent, hemoglobin 56 per cent. The patient sat propped up in bed quite dyspneic. The thoracotomy wound was healing. There was an enormous area of precordial dullness extending one and one-half fingers to the right in the fourth interspace and to the lateral chest wall on the left. No heart impulse could be seen or felt. The heart sounds were faint, no murmurs were heard. Edema was marked in both legs. The abdomen was distended. The liver margin was at the level of the umbilicus.

X-ray showed a water bottle shadow. On April 25, 250 c.c. of thick white pus were removed from the pericardium by paracentesis, with improvement of heart action. Pneumococci were reported in the pus. April 26, 175 c.c. of pus were removed and on April 27, 50 c.c. were obtained.

Phlebitis developed in the left leg with swelling and tenderness along the posterior tibial vein.

On April 30 the patient was referred for operation and under local anesthesia the sixth costal cartilage was resected and the pericardium drained. Although mechanical embarrassment of the heart was relieved the patient died of general sepsis May 4, 1931. No autopsy was obtained.

CASE III. Mr. H. O. S., white man, aged twenty-nine, was admitted to the Columbia Hospital December 4, 1931, having been sick since November 11 with pneumonia. His temperature was 102.5°F., pulse 103, respiration 24. A left non-encapulated empyema had been drained by thoracotomy on the day of admission. Pneumococci were found in the pus.

December 21, 1931 he was referred to me for surgical drainage after pus had been found in the pericardium by needle. A verticle incision was made along the left sternal margin and about 1 inch of the sixth costal cartilage was removed. The pericardium was opened and 4 to 6 ounces of purulent effusion escaped. Pneumococci were reported in the fluid. The wound was drained with a wick of folded rubber dam.

The temperature from 101.8°F. at the time of operation reached normal on the fourth day, after which it did not get above 99°F. until the sixteenth day when it reached 101°F. after 300 c.c. of whole blood had been given by transfusion. He grew gradually weaker and died January 13, 1932, twenty-three days after pericardiotomy.

On January 12 *x*-ray examination showed a shadow in the left chest in close proximity to the heart shadow. Pus was obtained by paracentesis but the patient was in extremis and thoracotomy was not attempted.

At autopsy the pericardium was found empty except for masses of adherent fibrin. An encapulated left empyema was found above and behind the pericardium.

CASE IV. T. R., white boy, aged twelve, was admitted to the Baptist Hospital January 12, 1933 after having been sick four weeks with a deep abscess of the thigh which had been incised. He later developed pneumonia. On admission his temperature was 104°F., pulse 130 and respiration 30, leucocytes 18,000 with 80 per cent polynuclears, hemoglobin 60 per cent. His urine contained albumin and casts. Physical examination showed an acutely ill emaciated boy with fair expansion on both sides but with râles over the right chest. The heart dullness was enlarged. A definite friction rub was heard over the base of the heart.

X-ray examination showed a large water bottle shadow filling much of the chest. There was also consolidation from unresolved pneumonia in the right upper lobe. Pericardial paracentesis was done the day of admission and staphylococci reported in the fluid.

On January 13, the day after admission, the sixth costal cartilage was resected and about 6 ounces of purulent effusion containing masses of fibrin were drained from the pericardial cavity. Before incision of the pericardium the left pleura was accidentally opened and was closed with catgut. The pericardium was drained with a rubber wick.

He improved after operation but continued septic. On January 18, 1933, by the closed method, a left empyema was drained. Staphylococci were cultured from the empyema pus from the pericardial pus and from the blood stream. He was at times extremely ill and had numerous septic exacerbations with high fever. He remained in the hospital until April 3, 1933, seventy-one days.

Examination March 15, 1934 shows a robust boy, a football player, with normal heart action, normal heart shadow and normal electrocardiographic record.

CONCLUSIONS

1. Suppurative pericarditis is not rare.
2. It is essentially a disease of children and of young adults.
3. Although marked by characteristic physical and roentgen findings, accumulating evidence proves that most cases are not recognized clinically, a serious indictment of the medical profession.
4. The condition is not diagnosed because it is not suspected and is not looked for.
5. Cases treated medically have a mortality of about 100 per cent.
6. The treatment is exclusively surgical.
7. Prompt pericardiotomy should materially reduce the present surgical mortality of 40 per cent.
8. Close cooperation of clinician, roentgenologist and surgeon at all times is essential.

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[For Discussion see p. 639]

CORONARY OCCLUSION SIMULATING AN ACUTE ABDOMINAL EMERGENCY

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A PATIENT was sent into the hospital with the story that about six hours previously while carrying a heavy load he experienced a sudden sharp pain in the epigastrium, in fact, he thought he had been shot, and collapsed immediately. His fellow workmen thought he was dying, but got him into an automobile and took him to his home, several miles distant over a bad road. By this time he had revived somewhat, but collapsed again while being taken out of the automobile. The family doctor saw him about two hours after the onset, at which time the patient was in moderate shock, with subnormal temperature, moderately rapid pulse, and complaining bitterly of pain in the epigastrium. The abdomen was everywhere boardlike. He was given a quarter grain of morphia hypodermically, followed in about an hour by a second similar dose. While the abdomen still remained rigid, the pain was somewhat relieved thereby. Inasmuch as he had a previous history of some indigestion over a period of several years, he was sent into the hospital by ambulance with a tentative diagnosis of ruptured ulcer. On admission there presented a well-nourished, heavily muscled man of about fifty in quite acute distress and moderate shock. All of his pain was referred to the epigastrium. The abdomen was quite rigid and boardlike from the umbilicus upward; below that level it was somewhat softer, but still very spastic. The point of most acute tenderness was along the costal margin just to the right of the midline. There had been some nausea, but no vomiting. The pulse was 104, of fair volume, regular in force and rhythm. Blood pressure 120/80; temperature 102.6°F. rectally. The white count was 19,100 with 93 per cent polymorphonuclears. Lungs clear on percussion and auscultation; normal heart sounds and cardiac dulness. Urine normal. A diagnosis of ruptured duodenal ulcer was made, but inasmuch as it had occurred on a presumably empty stomach, over five hours after a rather light lunch, and inasmuch as the rigidity of the lower abdomen appeared to be relaxing somewhat, and rectal examination was negative, it was thought that the hole had probably been sealed over by omentum, and it might be well to wait until the process had localized and walled off more securely in the upper abdomen; therefore,

it was decided to defer operation for the time being. Apparently this was good judgment, as the following morning the symptoms had subsided markedly, the rigidity being confined entirely to the upper abdomen, though the temperature, blood pressure, and pulse rate remained about the same. There was still marked tenderness on palpation in the right upper quadrant. Operation was again deferred, and the following day he was so much better that he wanted to get up. An *x*-ray taken to see if there was any evidence of free gas under the diaphragm was reported negative, with normal lung shadows and cardiac stripe. The spasticity of the upper recti had almost disappeared except in an area just below the costal margin on the right side. That night the nurse saw him at midnight and he was fine; twenty minutes later when she looked in again to see if he had gotten back to sleep, she found him gasping, and he died before the interne could be called. At autopsy the abdominal findings were entirely negative, a perfectly normal gall bladder, stomach, pylorus, and duodenum; no sign of any present or past trouble. The chest was otherwise, the pericardium being tremendously distended with fresh blood clot. The heart was compressed. On the posterior surface of the left ventricle, near its apex was a ragged hole, which would admit the tip of a finger, situated in the center of an area of softened, recently infarcted muscle about the size of a silver dollar. On dissecting out the coronary arteries many atheromatous placques were encountered, with scarred areas in the heart muscle indicative of previous small infarctions.

We at least had shown good judgment in deferring operation, though on an entirely wrong diagnosis and reasoning. We shuddered, and yet were at a loss, even looking back over the case, to see how we could have recognized the actual condition.

Not long thereafter, we were called in by a confrere, a prominent gastroenterologist, to operate on his uncle.

He had previously been a patient of ours, and this was his eighteenth admission to the hospital over a period of twelve years. He had originally had a carcinoma of the cheek, which had been treated elsewhere with *x*-ray and radium, resulting in an extensive slough of the soft parts and necrosis of the mandible. The previous admissions had been for operations on the jaw, and plastic repairs to the soft parts. During these years of observation and many admissions, there was some mention of mild indigestion at times, but nothing more. There had been numerous general anesthetics administered. The blood pressure readings ranged from 110 to 138 systolic, and 64 to 80 diastolic. The present attack, as described by his nephew, who had sent him into the hospital for immediate operation,

sounded typical of an acute gall-bladder attack. His tentative diagnosis was "acute cholecystitis, possibly perforated, with probable acute pancreatitis." I first saw the patient there, in the ether room. A rather hasty, superficial examination revealed a moderately thin, old looking man, suffering acutely, with a history of severe pain in the upper abdomen under the costal margin, of sudden onset, three days' duration, little if any let up. The abdomen was almost boardlike, but the greatest tenderness seemed to be in the right upper quadrant where there was a suggestion of an indefinite mass. Lungs were normal. There was a long systolic murmur at the base of the heart (this had been noted on previous admissions) but with no cardiac enlargement or evidence of decompensation. Temperature, 103.2°F.; pulse 100; respiration 22; blood pressure 125/80; white blood count, 13,600; polymorphonuclears 85 per cent. Operation was proceeded with immediately. On opening the peritoneal cavity a redundant loop of transverse colon, in the region of the hepatic flexure, packed with very large hard fecal boli, presented in the wound. After getting it out of the way, the gall bladder was found perfectly normal in appearance, with no palpable stones in it or any of the ducts. The head of the pancreas was slightly indurated; stomach and duodenum normal. The appendix had been previously removed, and there were a few omental adhesions beneath the McBurney scar; the cecum was quite bound down and contained numerous fecal boli. The small intestine was normal. The left kidney was larger than the right, but both felt normal. Numerous rather hard sclerotic placques were felt in the wall of the abdominal aorta. We overlooked the significance of this, completely. The fecaliths were broken up with some difficulty, and passed along to the descending colon and sigmoid, in so far as possible. The adherent omentum and cecum were freed, and the wound closed. The postoperative diagnosis was then "fecal impactions with intestinal obstruction." The patient continued to have very severe and constant pain in the abdomen, not limited to any definite point, accompanied by marked distention. Morphia and other sedatives gave little or no relief of pain, and enemata with pituitrin little or no relief of the distention. The second day after operation the same conditions persisted; a gastric lavage was returned dark colored, bile stained, and with a foul odor, though not typically fecal. Temperature, 98.4°F.; pulse 94; repiration 28. That evening the heart started fibrillating, which gave the first intimation of the correct diagnosis. An electrocardiograph, made the following morning, five days after the onset of the attack, was reported as follows:

"There is very slight depression of RT_1, and marked elevation of ST_3 and ST_2. The T waves are not inverted in lead 3 or lead 2 as yet. This tracing is characteristic of an infarction, possibly still active." There was little change in the general condition of the patient throughout that

day, and the following morning he died rather suddenly, of a rapid cardiac failure with pulmonary edema. During the entire postoperative course of three and one-half days, the abdominal distention could not be relieved, and the patient suffered excruciating pain in the abdomen, but never in the chest. At autopsy the pericardial sac contained hemorrhagic cloudy fluid. The heart was moderately enlarged. The lateral border of the left ventricle was covered with patchy fibrinous exudate. The wall of the left ventricle showed a sharply defined margin separating an acute myocarditis from a relatively normal myocardium. This area was typical of a recent infarct. In the left coronary artery about 1 cm. distal to the bifurcation of the anterior descending branch, there was a complete obliteration of the left coronary and the beginning of infarction. Both coronaries were full of arteriosclerotic placques and segments of definite constriction of the lumen of the vessels. The abdomen was negative except for marked distention of the bowel everywhere, but without evidence of any type of organic obstruction. Otherwise the autopsy findings were entirely negative. In this case we certainly did the patient no good by operation.

A few months later, when a very excellent medical man called us in to see an acute gall-bladder attack in a man who had been a patient of his for several years, this last experience was fresh enough in our mind to make us a little suspicious, inasmuch as the picture did not seem to be altogether typical. In spite of some pressure brought to bear for immediate operation, we held off. His story, in brief, was this:

Six days before admission to the hospital, which latter was when we first saw him, he began with a sore throat. At that time the temperature was 102.0°F.; blood pressure 128/85. Examination was negative except for red fauces, a few râles at both bases, and some tenderness in the right hypochondrium. This picture showed little change until the morning of admission when the patient had a sharp attack of pain in epigastrium, with no radiation. There had been slight dyspnea since the onset of the present illness; this morning there was also a little orthopnea. There was no precordial pain, or pain in the chest; no jaundice, clay colored, or dark stools. Frequent sour eructations of gas occurred. Physical examination revealed a heavy set man of fifty-nine years. The throat was now only slightly red. Respirations were somewhat rapid, 32, regular, rather shallow, almost wholly costal. Numerous fine and medium râles were noted at both bases. All heart sounds were distant; no murmurs; apex impulse not palpable. To percussion the outline was normal. The pulse was regular, rather soft, rate 120; blood pressure 120/88. Abdominal muscles were held rigidly.

The liver edge was apparently two finger breadths below the costal margin on the right, and slightly tender. The most marked tenderness was just below the xyphoid in the midline. The lower abdomen was more relaxed. Soon after admission he began to have rather marked attacks of dyspnea, with pulse running up to 140 or 150, but perfectly regular. The pain rapidly disappeared, though some tenderness below the xyphoid persisted. The third night after admission his blood pressure rapidly dropped until unobtainable, and so remained for thirty-six hours, although during this time the patient was perfectly conscious and oriented, and while quite dyspneic, complained of no pain whatever. The heart sounds were barely audible, rate apparently about 120 to 140. Temperature ranged from 98.0° to 100.2°F.; respiration from 28 to 40. There was marked abdominal distention, but without pain or tenderness. The blood pressure gradually picked up, and he developed general dependent edema, with pericardial and pleural effusions, and ascites. Pericardial and pleural friction rubs could be heard at times. The chest was tapped three times, and the first time 1000 cc. bloody fluid were removed; 1500 c.c. straw colored the second; and 1600 c.c. straw colored the third. He was in the hospital, all told, about three months. Since discharge he has had several attacks of abdominal pain, but none severe enough to warrant his return to the hospital. Most of these have been in the epigastrium, with marked tenderness and muscular rigidity for awhile. Two attacks were low enough in the right lower quadrant to raise the question of appendiceal origin. In every instance, except the first, the blood pressure showed a drop of 25 to 40 points within a few hours of the onset of pain. In no attack has the pain been referred to the precordium or down the left arm. Every attack has shown a moderate to high leucocytosis, and moderate temperature rise. The patient is at present a semi-invalid, but able to be at his office at times.

The similarity of recurring gall-bladder attacks to anginal seizures has been recognized for some time, together with the difficulty, frequently, of differentiating between them. This problem is further complicated by the now fairly well-established connection between chronic gall-bladder disease and myocardial damage. We feel, however, that not nearly enough emphasis is placed upon the ability of a purely cardiac lesion to simulate an acute abdominal emergency so closely as to make it almost, if not entirely, impossible accurately to distinguish between them. It certainly is not necessary to point out to a group such as this, the danger and disastrous results

of ill-advised operation due to a faulty differentiation between these conditions. It is for the purpose of again calling attention to this difficult problem of diagnosis, that we present this paper. We feel that it may not be ill-timed or amiss, inasmuch as the insurance companies are reporting a tremendous increase in the incidence of coronary accidents during the past two years.

In a review of some forty odd articles in the literature, contributions by Barnes, Brown, Halsted and Bauer, and Faulkner, Marble, and White seem particularly pertinent. The last named is especially so, in that it tabulates the points of differential diagnosis in 30 cases of coronary occlusion and 30 cases of cholecystitis; we draw extensively upon it. We will attempt no further reference to or summarization of the literature in the short time at our disposal.

Similarly in the matter of referred pain, while a great deal of work has been done, and much written, dealing with the subject from anatomical, experimental, and theoretical standpoints, we can not, for obvious reasons, not the least of which is our own ignorance of the subject, enter into any discussion at a time such as this. Suffice it to say that fibers of the vagus and sympathetic system supply both the heart and upper abdomen, and that they exit from the spinal cord within one to four segments of each other.

In an effort to determine whether the site of the coronary occlusion played any part in the reference of the pain to the abdomen, we were able to pick out 5 case records at the Johns Hopkins Hospital, and two at the Union Memorial Hospital, all from patients in whom the pain had been purely abdominal, and in whom subsequent autopsy revealed definite myocardial infarction and negative upper abdominal findings. Five of these had infarcts in the anterior wall or apex of the left ventricle, 4 from occlusion of the anterior descending branch of the left coronary, and one from occlusion of the circumflex branch of the left coronary; 2 had infarcts in the posterior wall of the left ventricle with occlusion of the right coronary

artery. In 2 other cases which did not come to autopsy, the electrocardiagraphic interpretation in one was occlusion of the right coronary artery; in the other, the left. From this it would seem that neither the artery occluded nor the site of that occlusion has any controlling influence over the occurrence of abdominal pain in coronary accidents.

In 5 of these cases, electrocardiograms showed characteristic changes of the RT segments and the T waves. It must be borne in mind, however, that the diagnostic value of the electrocardiogram is comparatively slight when the tracing is made shortly after the onset of the attack, and that a certain amount of time, often as much as several days, must elapse before the typical curve is obtained. In one of our cases this fact is so well illustrated that we will quote the reports of four successive tracings. The first was made on the third day after onset of symptoms. "Normal Sinus rhythm. Changes in the RS T segment indicate myocardial disease and, while not pathognomonic, are entirely compatible with a recent coronary occlusion." Three days later we are told, "The changes which have occurred in the RS T segment since the previous record are strong evidence in favor of a recent myocardial infarction. Record is of the T_1 type." Then after four more days, ten days from the time of onset: "The record is fairly characteristic of the T_1 type seen after thrombosis of the anterior descending branch of the left coronary artery, with infarction of the anterior apical portion of the left ventricle." Finally, one week later, "Since the previous tracing there has been a slight shift of the RS T segment toward a normal appearance. The coronary T waves are conspicuous and the notching of the QRS in Lead 1 suggests myocardial damage." Like "laboratory methods of precision" in a good many other instances, the electrocardiogram furnishes valuable confirmatory evidence at times, but can not be depended upon solely for a diagnosis, particularly in the early stages of the disease when its recognition is most important, and differentiation most perplexing.

Another sign which we might expect to be a real diagnostic aid, and which we usually associate with a coronary thrombosis, is a sudden, marked fall in the blood pressure. This, however, is not nearly so helpful as one might imagine at first thought. Many patients in whom we are called upon to make the diagnosis, are seen for the first time after the onset of the acute symptoms. Therefore we have no way of knowing what their blood pressure normally registers, for comparison with the reading which we may obtain at that time—witness our first case referred to. Consequently, unless there is a markedly abnormally low pressure, there would be no reason to have our suspicions aroused on that score. And further, there is a group of cases in which there is no appreciable initial drop, for example, the second and third cases which we cited, even though we are in possession of a base line for comparison with the present reading. The doctrine that coronary occlusion is always accompanied by an immediate drop in blood pressure is far from true; the drop is often noted only after several hours or even days, and sometimes does not occur at all. When noted, it is a great aid in the correct diagnosis; when absent, it may be only misleading.

Upon what are we going to base our diagnosis, then, for we must have something to work on immediately, while trying to decide whether this particular case is a surgical emergency, warranting immediate operation? If it is a perforated ulcer, either gastric or duodenal, it almost surely demands such urgency; if a ruptured gall bladder, or one with associated acute pancreatitis, speedy intervention is just as necessary; and even in the event of a simple acute gall-bladder attack, the trend is more and more toward applying the same principles as have long obtained when dealing with the appendix, namely, prompt removal.

Unfortunately, so far as we can determine, there is no sure way of making the differential diagnosis between any of the preceding conditions, and the comparatively rare case of coronary thrombosis with purely abdominal symptoms. But

there are certain considerations which may help toward a proper solution of the dilemma. First, there are two general factors which should be taken into account, age and sex. In the cases analyzed by Faulkner, Marble, and White, the average age of the gall-bladder group was forty-seven and two-tenths years, with extremes of twenty-three and sixty-five years; the average age of the coronary occlusion group was fifty-eight years, with extremes of forty-two and seventy-six. Our own cases of the latter were all in the fifties, with an average of fifty-four and seven-tenths years. Apparently, at least 65 per cent of coronary occlusions will occur after the fiftieth year, though the incidence in younger individuals does seem to be increasing. Now as to sex, gall-bladder disease is unquestionably more common in females, some figures putting the ratio as high as four to one. On the other hand, coronary occlusion is much more frequent in males, the estimated ratio running as high as four and one-half to one. All of our cases of the latter were males.

Next, a most essential diagnostic aid is a very thorough and carefully taken history, not only of the present illness, but, in minute detail, the past history as well. Recognizing the fact that in many cases chronic gall-bladder disease may masquerade for years as mild angina, and vice versa, still it is not infrequently possible, by very careful questioning, to get a definite lead in the right direction. Has the discomfort been precordial; has there been a feeling of construction of the chest; has it been associated with any form of activity; has there been any dyspnea; have there been any abnormal sensations associated with the heart beat, such as tachycardia or irregularity? On the other hand, has there been indigestion characterized by eructations and flatulency; was the discomfort related to the ingestion of food, and if so, any particular kinds of food; have the attacks ever come on while at rest, as when in bed at night; has there been any periodicity, as related to meal time, time of day, or season of year; have there been attacks of severe pain, other than the present one?

Affirmative findings in the first group point toward cardiac origin, in the latter group, toward abdominal. Certainly the longer the history, and the more frequent, prolonged, and severe the attacks of discomfort or pain, the less apt is it to be cardiac in origin. However, one of our patients had been under observation and treatment for over seven years by an eminent gastroenterologist for chronic cholecystitis, which autopsy proved he did not have, though there were numerous small fibrotic scars scattered throughout the heart muscle.

Finally, a close scrutiny and judicious evaluation of the story and symptoms of the present attack, correlated with the physical and laboratory findings. The pain in coronary occlusion (we are referring, of course, only to that small group with abdominal symptoms) may be absolutely localized in the gall-bladder region, more rarely lower down in the right lower quadrant, but most frequently possibly in the mid-epigastrium, nearly in the midline below the xyphoid. It rarely, if ever, radiates through to the back, as the pain of a distended gall bladder tends to do. The point of maximum tenderness on pressure is also apt to be more nearly in the midline than with an acute gall-bladder attack. This may be in line with the sudden enlargement of the liver seen in the rare cases of infarction of the right side of the heart, and be dependent upon stretching of the capsule of the liver, as well as referred cardiac pain. Also, while we are considering the liver, although jaundice is not a common finding in coronary occlusion, there have been a number of cases reported, the jaundice, in some instances, having been quite marked, so that its presence, while suggestive, is not in any way pathognomonic of gall-bladder disease. Dyspnea, palpitation, or the sensation of irregularity of heart rhythm is much more common with coronary occlusion, though it may occur in other conditions. Signs of cardiac failure such as cyanosis, weakened heart sounds, râles at the lung bases, and sudden drop in blood pressure, are obviously in favor of coronary occlusion, but are often rather late manifestations. Still, even these may be associated with the

shock seen in acute pancreatitis. A precordial friction rub is pathognomonic, but can be heard in a relatively small percentage of cases, and then usually late. The temperature and pulse rate seem to have a tendency to be slightly higher in coronary occlusion, as is also the case with the white blood count. As has already been mentioned, any help from electrocardiography is apt to be too late to be of use in making the immediate diagnosis, but should be utilized, if available. It will eventually show changes characteristic of cardiac infarction in probably 90 per cent of cases.

In summary and conclusion, we present in this paper no new disease or diagnostic principle, but merely call your attention anew to a pseudo-surgical emergency which can offer as nice a problem in differential diagnosis, for the exercise of sound judgment, as may ever face any of us.

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DISCUSSION ON PAPERS OF DRS. BUNCH AND FINNEY, JR. AND MOHR

Dr. Nathan Winslow, Baltimore, Md.: We feel that many of these cases are being overlooked by the profession. Certainly it would seem so when members on the staff of the University of Maryland have operated on 15 cases during the past ten years. Of these 15, 9 patients, or 60 per cent, recovered. Shipley and I have reported 10 of these cases in detail.* Six, or 60 per cent, of the patients in this group recovered, so there seems to be

* *Arch. Surg.*, (Sept.) 1927.

no improvement, as yet, in the operative results. As none of these patients would have lived, we feel that a salvage of a little more than 50 per cent of them amply demonstrates the advantages of the operative over other kinds of treatment for pyopericardium.

Purulent pericarditis is an abscess and, like any other abscess should be treated by incision and drainage. Too much cannot be said in favor of early operation, but late operation should not be denied these patients. The best approach is the costoxiphoid route with resection of the left fifth, sixth and seventh costal cartilages, and, if more room be needed, a piece of the adjoining sterı um may also be removed.

There are three chief factors which govern the prognosis: (1) the time of the operation; (2) the type of the organism, and (3) the original condition of which the pyopericardium is a complication. Obviously, suppurative processes in other parts of the body, unless detected early and promptly remedied, render the prognosis less favorable. Most of these patients (80 per cent) are under thirty years of age; 70 per cent are males and 30 per cent females. Diagnostic aspiration is rarely indicated. The diagnosis should be made on the clinical signs and the characteristic *x*-ray shadow. When the pericardium contains fluid, the heart is usually found lying against the anterior wall of the sac. This may give a misleading nearness and clearness to the cardiac sounds. Troublesome postoperative adhesive pericarditis does not occur as often as is generally supposed.

Dr. Bunch has done a real service by calling attention to these cases, for wherever pneumonia is prevalent, there also are to be encountered, at times, patients suffering with purulent pericarditis.

Dr. Goodrich B. Rhodes, New York, N. Y.: In the beginning of this lesion the pus is localized in two pockets before the whole of the pericardium is filled. One of these is in the aortic region and the other is above the diaphragm. Both of these pockets are posterior to the heart. This early formation of pus can be diagnosed by *x*-ray and can easily be misinterpreted as a dilatation or an aneurysm of the aorta.

As a result of the posterior location of the pus formation the heart is pushed forward against the chest wall by the increasing exudate. I have studied reports of 118 operated cases, and in all cases in which the position of the heart was stated it was found to be anterior. This I consider a very important finding, for in at least 2 reported cases the heart has been punctured with fatal results through ignorance of this pathological anatomy.

Second: the anatomy of the pericardial reflection of the pleura and its relationship to the sternum is very vague in all our anatomical textbooks. And for a very good reason. It is very indefinite and varies within very wide limits. In order to study this at first hand I requested the dissecting room students to make sketches of these relationships in all the bodies.

These showed such wide variations that the conclusion is forced upon me, that in no given case can we insert a needle into the pericardium and be certain of not entering the pleura. I have seen this happen in one case, causing an empyema, which determined the death of the patient.

For these two reasons: the forward position of the heart and the uncertainty of the location of the pleura, I am opposed to paracentesis. No reported case has been cured by repeated aspirations, and as a diagnostic measure it is unnecessary. The diagnosis can be made by the physical signs, circulatory disturbance and *x*-ray. No case treated medically has recovered and this is distinctly a surgical disease. The sooner this is recognized by internal medical men and the earlier the patients are turned over to us the better will be the results.

Dr. I. A. Bigger, Richmond, Va.: Dr. Bunch has pointed out that the mortality rate in patients with suppurative pericarditis treated conservatively approaches 100 per cent, whereas in those given adequate drainage approximately 50 per cent recover. Relatively few patients with suppurative pericarditis die from the pericarditis, if the pericardium is adequately drained, but a considerable number of them will die from associated conditions, as for example, abscesses in the liver, spleen, kidneys, etc. The results from operative treatment therefore are actually better than the mortality statistics would seem to indicate.

I have operated upon 4 patients with suppurative pericarditis, with 2 recoveries and 2 deaths. The first patient had an empyema of the right pleural cavity with an associated suppurative pericarditis. The pericardium was aspirated and then drained. The patient developed an empyema of the left pleural cavity which was later drained. He eventually made a complete recovery.

The second patient had an acute osteomyelitis of the femur and a Staphylococcus aureus septicemia. She developed evidence of suppurative pericarditis about forty-eight hours after the onset of the osteomyelitis and the pericardium was drained as soon as the diagnosis was made. She eventually made a good recovery.

The third patient, a white child twenty months of age, had an osteomyelitis of the distal phalanx of the left ring finger, which was not adequately treated. He apparently developed a septicemia and then a suppurative pericarditis. When first examined this child was desperately sick and was operated upon under local anesthesia a short time after admission to the hospital. He showed considerable improvement for about one week, then became worse, and died about two weeks after the pericardium was drained. Autopsy showed the pericardium to be quite clean, containing no actual pus. There were Staphylococcus aureus abscesses in the lungs, liver, and spleen, which had obviously caused the patient's death.

The fourth patient, a white boy fourteen years of age, had a total pneumonectomy done for a carcinoma of the left lower lobe bronchus. He had an empyema at the time of the pneumonectomy and a few days later developed evidence of suppurative pericarditis. He was operated upon and the pericardium drained, with a resulting marked improvement in his general condition for about one week. He then developed enlargement of the liver, ascites, and edema of the trunk from the costal margin down and of the lower extremities. The pericardium was markedly thickened, so the anterior portion of it was excised and the posterior portion of the pericardial sac explored for sacculated fluid but none was found. He seemed to show temporary improvement from this procedure but after a few days he became rapidly worse and died. Autopsy revealed a marked constriction of the inferior vena cava at the point of its entrance into the pericardial sac. There was also a septic hemorrhagic infarction with bronchial pneumonia in the right lung.

Dr. Joseph A. Danna, New Orleans, La.: Most of the deaths in suppurative pericarditis are due to associated conditions, or to the original condition to which the pericarditis is secondary.

Since a suppurative pericarditis presents the same conditions as those found in empyema, both conditions it seems to me should respond to the same treatment. These patients are very sick and the less we do to them the better they stand it. I should like to suggest, therefore, their treatment by aspiration and air replacement which has given me and many others who have used it such good results in the treatment of empyema.

There are three reasons why I think it should be better than open drainage. One is that it causes less shock and less trauma to the patient. Second, if you empty the pericardial cavity entirely of fluid, the pericardium will have an opportunity during the two or three weeks it would take the air to be absorbed to resume to a great extent its normal state once more and the formation of adhesions would be minimized. There is another important reason and that is this, that with a heart surrounded by air an *x*-ray picture will show very clearly whether there is additional abscess formation, and the patients who would otherwise die because an additional pocket of pus is undiscovered and undiagnosed will be caught and this pocket can be drained.

Dr. Frank P. Strickler, Louisville, Ky.: I wish to call attention to the blood pressure in coronary occlusion. We know that coronary occlusion occurs from fifty to sixty years of age. We know that the blood pressure drops materially in these cases, also in shock. In cases of shock there has been no damage done to the heart muscles, and the blood pressure will rise when shock treatment is instituted.

In the cases of coronary occlusion the heart muscles and vessels themselves have been damaged. This is followed by a drop in the blood pressure, then a gradual rise to around 100 mm. Hg and the blood pressure remains stationary at this point.

I have seen several cases of coronary occlusion which had a surgical aspect, and have discussed this subject with some of my heart specialist friends, and they appear to be of the same opinion as myself. I feel if one will carefully watch the blood pressure readings, one will get an earlier line on these patients than we can hope to get with electrocardiograph tracings. I think that most any well-trained doctor can take blood pressure readings with a reasonable degree of accuracy, and thereby make a much earlier diagnosis.

Dr. K. H. Aynesworth, Waco, Texas: I wish to discuss the feature of pain in connection with Dr. Finney's paper. In 1914 a patient was brought in with symptoms of an acute emergency. I operated and found the viscera perfectly normal, which was a great shock to me. After operation I studied the patient as I should have studied him before operation. It was an anginal pain and I did not understand coronary occlusion then as I do now. I found the gall bladder normal, but I had to do something. Fearing that it might be gall-bladder pain, a drainage tube was put in the gall bladder. Soon afterward I had a letter from a doctor in another town asking for my records and saying they wished to operate. I telegraphed him not to operate for it was a heart condition. That was twenty years ago. From the internists I have not had the help that I wanted, but if we take the clinical signs the pain is of the same kind that often occurs in acute upper abdominal conditions. The pain is splanchnic, which is diffuse. It is not, in my experience, paroxysmal. We know that, embryologically, the heart is supplied with the same nerves as the intestines. The vagus supplies the heart and the small intestines; also, we know there is a splanchnic sympathetic nervous connection to both the heart and the gastrointestinal tract down to the large bowel and in these structures the pain is always diffuse. It may be in the hypochondrium or in the hypogastrium or anywhere, but it is diffuse and more or less paroxysmal, but in the heart it is unlikely that we will have the same type of pain as in the intestine. The nerves in the heart go to the ganglia of the central nervous system and connect with the motor nerves that go to the chest wall. That is why we have a sense of compression in the chest and the pain is very diffuse; it may be felt in the chest, in the neck and in the upper abdomen. It is characterized by more or less continuous pain in the heart and gives the symptom of excruciating pain in the chest which is never present in gall-bladder difficulties.

Dr. Finney spoke of tenderness. In my experience there is no tenderness in the abdominal wall and that is one of the characteristic symptoms of early coronary pain. The abscess of tenderness locally in the epigastrium is characteristic of heart pain and is just the opposite condition found in any pain due to perforation of the gall bladder, the stomach, or exudate in the abdominal cavity. On these symptoms alone, a continuous type of pain, the diffuse type of pain, and the absence of tenderness, the diagnosis of coronary pain can be made. In my experience the pain has been so diffuse that it could not be due to inflammatory lesions. Another thing is that the patient is not quiet, as in abdominal conditions, but very restless.

Dr. Roy D. McClure, Detroit, Mich.: Many cases of thrombosis and embolism, especially of the coronary vessels, can be avoided. From personal experience we know that an attack often starts after strenuous exercise, and I never miss an opportunity of preaching to my friends that they should not take needlessly strenuous exercise after the age of forty, when so many develop coronary trouble. I feel that in the treatment a certain amount of quietness is necessary, but it is very nice to have the confidence of the patients and make them feel that they have a chance for recovery after they have given up hope.

Dr. Rudolph Matas, New Orleans, La.: Dr. Finney has done well to direct attention to a pitfall in diagnosis which is easy to fall into when patients are brought to an emergency clinic in a state of shock or collapse with no adequate history, with signs and symptoms pointing to an acute perforative or occlusive abdominal lesion which calls for immediate exploration. It is only after the negative results of the exploration or the revelations of a postmortem brings to light the real seat of the lesion in the heart, that we realize how closely and deceptively an atypical angina of the celiac type can simulate an acute subdiaphragmatic lesion.

On the other hand, the error may be reversed when unexpected fatalities, occurring in the course of abdominal disease, and especially after operations for acute abdominal lesions, are attributed to heart failure. Now that we know so much more about coronary disease and thrombosis than formerly, the tendency is to transfer the responsibility for sudden postoperative deaths from the abdomen to the heart. Though this may be correct in most cases, the diagnosis of death by coronary thrombosis is often an assumption which, when not verified by necropsy, is subject to abuse as a convenient cover for postoperative complications that lie below the diaphragm.

The number of cases of purulent pericarditis which Dr. Bunch has been called upon to operate is much larger than that which falls to the lot of most surgeons. Though the reported cases of tapping or paracentesis of the pericardium are numerous enough in the literature, the cases of peri-

cardiotomy for purulent or other effusions are comparatively rare, and usually stand out prominently in hospital statistics when they are performed.

My own personal experience in over fifty years in the surgical service of the Charity Hospital of New Orleans and other institutions does not exceed, all told, 5 cases in which I have been called to operate for pericardial effusions, including one hemopericardium caused by heart wound. Two of these patients died and 3 made operative recoveries, but one of these died ultimately from a coincident endocarditis. One of these, the first case, in 1886, was an attempted aspiration which I had to transform into a pericardiotomy because of the bloody appearance of the serum. All the rest were pericardiotomies for purulent or seropurulent accumulation. The comparative rarity of pericarditis is shown in the statistics of the Charity Hospital. The annual reports of this institution from January 1, 1930, to July 1, 1934, show that only 30 cases of pericarditis were so diagnosed during that period, and of these, 25 were classified as acute pericarditis. During that period 223,657 patients were admitted to the hospital, which would make the incidence of the cases specifically classified as pericarditis, amount to about one case of pericarditis to 7500 admissions. What is more significant is that the surgical records for the same period show no pericardiotomies or open operations for acute pericarditis. Three cases were aspirated, with 2 deaths, none of which were purulent. That this incidence entirely underestimates the frequency of pericarditis is well shown by the fact that 4–7 per cent of all bodies that come to autopsy show evidence or relics of some past or old pericarditis. It is surprising, however, that despite this frequent cadaveric incidence, the cases that develop purulent pericarditis are relatively few. The Charity Hospital records for the time mentioned show that there were just 4 cases of purulent pericarditis, and these were only recognized as such at autopsy, all occurring as terminal complications of general infections, chiefly pneumonic. The statistics of pericardiotomy for *purulent* effusions which I once compiled from various sources covering the period from 1900 to 1927 amounted in all to 105 reported cases with a mortality exceeding 53 per cent. The 30 cases of pericarditis reported at the Charity Hospital just mentioned, yielded a mortality of 50 per cent without surgical intervention (100 per cent in 4 terminal purulent cases). There can be no question that many cases of acute pericarditis are overlooked when they appear as intercurrent or terminal complications in the pneumonic, influenzal, streptococcal, pyococcal and rheumatic infections. Even when recognized, the patients often die before the effusion has had time to develop signs of pericardial distention—and heart compression. In the struggle for survival from an overwhelming general infection, the patients are scarcely fit for the radiologic examinations that have so much simplified the diagnosis of the

effusions. When they do survive with a residual pericardial accumulation, whether purulent or otherwise, the patients are left so weak and exhausted that an operation, which might be relatively simple and safe under other circumstances, becomes one of grave consequence.

When the diagnosis of a pericarditis with effusion, whether serous or purulent, is established, the procedure to be adopted for its evacuation and permanent cure presents itself as a worrisome problem. Is it pus or sterile serum that we are dealing with? is the first question. To decide this question, a preliminary exploration is necessary. Under these circumstances, the temptation to tap or puncture the pericardial sac with a trocar or aspirating needle is very great, but the risk of wounding the heart and of infecting the pleura is no idle fear, and we hesitate. The history of paracentesis pericardii when spread over the chest and precordium is like a map plotted with the historic names of the innumerable surgeons who have sought spots where a needle or trocar could be thrust into the pericardium without fear of injuring the great vessels, the pleura, and the heart itself. The great number and variety of these so-called points of election is the best proof of their fallibility and unreliability. The "triangle of safety" which the Russian surgeon Voinitsch described so accurately nearly forty years ago, in the left lowest chondrosternal angle, is the nearest approach to the pericardium in a pleura-free mediastinal space.

Despite the fact, as Dr. Rhodes has remarked and I have confirmed by numerous dissections, that even this space is often overlapped and encroached upon by the left pleura, it is undoubtedly the best area to work in when pleural complications are to be avoided. The chief danger of paracentesis lies, not so much in the pleura, as in the liability to puncture or penetrate the thin right ventricle which, in large effusions, is pushed forward against the anterior pericardial wall, where it lies directly in the path of the needle. While hundreds of pericardial punctures have been made without accident, this danger is real, as attested by numerous instances in which the heart itself has been tapped or the coronary vessels injured. In view of the uncertainties that attend exploratory punctures, I have in all my cases perferred to expose the pericardium by open dissection carried through an oblique line along the left edge of the sternum and base of the xyphoid cartilage, resecting as much of the sixth or seventh costal cartilages at the sternal juncture as would permit digital palpation and exposure of the sac. A small incision is then made between forceps. In purulent cases, the incision is enlarged and the edges of the pericardium are sutured to the inverted skin or fascia, thus converting the operation into a pericardiostomy. In all cases, a soft catheter or the bulbous tip of a long suction apparatus is introduced into the posterior recesses of the pericardial sac where the bulk of the fluid usually accumulates in greatest quantity.

In all my operations I availed myself of local anesthesia, aided by preanesthetic sedation with morphia, etc.

There is much that could be said regarding controverted questions of drainage, after-treatment and the modifications of technique that are required in special cases, but this is beyond the scope and purpose of my discussion, which is chiefly to direct attention to the contradictions of clinical and postmortem evidence in regard to the incidence of pericarditis and to emphasize my conviction of the far greater safety of open pericardiotomies over the so-called simple methods of exploratory or therapeutic paracentesis. Furthermore, the points at issue and details of technique have been fully discussed in my previous publications, which in so far as pericardial and cardiac surgery are concerned, began as far back as the New Orleans meeting of the Association in 1907.*

Dr. George H. Bunch (*Closing*): I hold no brief for therapeutic paracentesis. The diagnoses in my cases were all confirmed by the needle before I saw them, and I can very well appreciate the difficulties under which the internist labors, as was shown in the last picture, congenital heart. This patient had a large shadow, bronchitis, a high leucocyte count and fever. Certainly, if the clinician is denied the use of the needle in diagnosis fewer cases of purulent pericarditis will be recognized and fewer cases will be operated upon.

Dr. J. M. T. Finney, Jr. (*Closing*): The point Dr. Strickler makes about the initial drop in blood pressure, then the rise, and then maintenance of level not as high as the original is quite clear and a real help if we know what the original blood pressure was; unfortunately we frequently get our first record only after the onset of trouble. But it does not always hold true for in one of our cases the blood pressure was exactly the mean of the pressure that had been maintained over a period of twelve years and eighteen hospital admissions.

I thank Dr. Aynesworth for what he said, but have to disagree a little about the tenderness, for in several cases I have seen there has been just as acute tenderness with muscular rigidity as one would get with a true intra-abdominal lesion. I think there is another possible means of differentiation there, and that is that the point of tenderness is apt to be more nearly in the midline under the xyphoid than to the right where we get most of the tenderness in ruptured ulcers and acute gall-bladder pathology.

* In revising the stenographer's copy of this discussion I have taken the liberty to add a number of facts and statements which I had prepared for the meeting, but which I did not read because of the limited time at my command. With the kind permission of the secretary, I have made these changes, which add to the matter but do not alter the gist or purport of my original remarks.

POSTOPERATIVE THROMBOSIS AND EMBOLISM*

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THE etiological factors preceding thrombosis and embolism are complex. No single factor is probably responsible for this distressing postoperative surgical calamity.

In a previous communication[1] published by two of us (Bancroft and Stanley-Brown) we discussed the physical forces that are generally acknowledged to be the most important, viz: dehydration, venous stasis, infection and trauma, and stated that we thought there was, in addition, a biochemical factor either inherent in an individual or induced by the operative trauma that renders the blood of a patient more coagulable. We reported our study of the blood clotting elements and showed how, by quantitative tests for prothrombin, fibrinogen, antithrombin and by doing platelet counts and platelet disintegration time, we could group patients' bloods into three types (those who clot normally, those with bleeding tendencies and those with clotting tendencies) and suggested a clotting index. This work was carried on for four years, at the end of which time we felt, as clinicians, that we had reached our limit and that we needed a research chemist to aid in our work. As a result, Quick joined us, devoting his entire time to this problem from the laboratory side.

Quick reviewed over a thousand articles in the literature and started by throwing out all of our previous tests, with the

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The early part of this work was carried out through the generosity of Mr. Charles Frueauff of New York and the latter part through the generosity of Mrs. Dudley Blossom, of Cleveland, Ohio.

[1] *Surg. Gynec. Obst.*, 54: (June) 1932.

idea that he would approach this subject from an impartial point of view. He repeated many of the experiments of other workers in this field, corroborating some and disproving others. He has simplified our concept of blood clotting and has revised our old tests and made them more practical for routine laboratory use. Before going into his work more fully, let us review the steps he took in arriving at his conclusions.

A study of the individual factors in blood clotting revealed the following data: There are four main elements, viz: fibrinogen, calcium prothrombin and thromboplastin.

Fibrinogen. It is normally present in blood plasma to the extent of 0.3 to 0.75 gm. per 100 c.c. of blood. In infections it is apt to be high, whereas in certain profound liver disturbances low concentrations have been found. Linton, however, in a small series of severe jaundice cases of various types actually found high values. Experimental as well as clinical evidence seems to indicate that the concentration of fibrinogen within wide limits has little influence on clotting time. We have found that by adding purified fibrinogen to plasma, no change in clotting time occurred. Likewise dilution of plasma with a resulting reduction of the fibrinogen to one-fifth of its original concentration has no influence on the speed of clotting, as we have shown in a recent study. The rôle of fibrinogen in clotting is passive and it seems to have no direct influence on clotting. It is of course conceivable that the concentration of fibrinogen can be reduced to such an extent that no clot at all is formed or if formed would lack strength and firmness. It is also reasonable to assume that any condition which brings about a concentration of the blood, such as dehydration, may show an abnormally high fibrinogen. Since such blood has a high viscosity and is therefore more subject to venous stasis, a condition favoring intravascular clotting, a high fibrinogen may indirectly indicate the possible danger of thrombosis.

Calcium. The indispensability of calcium in the clotting process has led to the belief that in certain hemorrhagic diseases a deficiency of this element in the blood may be present. At

the onset it must be emphasized that even in the most marked hypocalcemia of parathyroidprevia, no definite alteration of blood clotting can be demonstrated, and likewise the high blood calcium in hyperparathyroidism does not bring about any marked change in the coagulation time, and even in cases in which the blood calcium is 100 per cent above normal no intravascular clotting has been reported. Clinically, normal blood calcium is found in hemophilia, and in the hemorrhagic diathesis of obstructive jaundice. No elevation of blood calcium has been demonstrated in spontaneous thrombosis. Whether the calcium concentration of the blood is optimal for coagulation is difficult to decide.

Prothrombin. Although a few investigators have doubted or even denied the actual existence of such a substance as prothrombin, there is overwhelming evidence that there exists in blood an agent which is the precursor of thrombin. It has the properties of a proenzyme, and therefore its isolation in pure form at present offers insurmountable difficulties. Its determination must therefore depend on first converting it to thrombin, which can be determined from the clotting time. The prothrombin is associated with the globulin fraction of the blood and appears to be limited to the blood. It varies in both bleeding and clotting diseases along a moderate range.

Thromboplastin. Much confusion concerning this substance still exists and it is still called by a variety of names such as tissue extract, tissue juice, tissue fibrinogen, cytocyme, cephalin and the like. The substance appears to be a protein-phospholipoid which possesses the power to convert in the presence of calcium prothrombin into thrombin. Unlike prothrombin, which appears to be limited to blood plasma, thromboplastin is widely distributed in the body. In circulating blood it is locked up in the platelets, but in shed blood it is liberated when the platelets disintegrate. The exact reason for the platelet lysis is not known. While platelets constitute the immediate and probably the most important source of thromboplastin, it is probable that all cellular structures contain

this substance and liberate it when injured. The lungs, brain, thymus, testes and other tissues serve as sources for this material.

The original premise on which we started the concept of blood clotting is as follows:

$$\left\{\begin{matrix}\text{Prothrombin} \\ + \\ \text{Fibrinogen}\end{matrix}\right\} + \text{Platelets} \rightleftharpoons \text{Antithrombin}$$

Expressing this reaction in terms of the law of mass action we have

$$\frac{(\text{Prothrombin})\ (\text{Fibrinogen})}{\text{Antithrombin}} = 0.6$$

Introducing normal values for these substances

Prothrombin Index	= 1
Fibrinogen	= 0.5%
Platelets	= 200,000
Platelet Lysis	= 50% in 1 hour
Active Platelets	= 100,000
Antithrombin Index	= 1.0

The normal index of clotting function of blood is therefore 0.6 ± 0.2. Values over 1.0 indicate a marked tendency to clot and values below 0.3 indicate a marked tendency to bleed.

This concept has changed somewhat and we now believe the following to be a more practical idea of clotting:

Blood Clotting Factors in Normal Blood

Prothrombin + Platelets + Ca + Fibrinogen

Blood Clotting Factors in Blood Preceding Thrombosis

Prothrombin + Thrombokinase (from Platelet Disintegration) + Ca + Fibrinogen.

Resulting in Blood Clotting

Prothrombin + Ca + Thrombokinase = Thrombin

Fibrinogen + Thrombin = Fibrin + Thrombin

As the rate of clotting is dependent upon the thrombin concentration in the blood it has been considered, in this work, as the basis for developing a practical quantitative method of determining blood coagulation time.

TEST FOR PLASMA CLOTTING TIME

1. Put 1 c.c. of M/10 sodium oxalate in test tube and add 9 c.c. of blood. Let it stand until red cells settle out (not longer than one hour).

2. Put 0.1 c.c. of plasma in each of 2 tubes. To tube No. 1 add 0.1 c.c. M/40 calcium chloride. To tube No. 2 add 0.2 c.c. M/40 calcium chloride. Shake and put in water bath heated to 37°C. Time exactly and record shortest time required for the formation of a solid clot. Normal clotting time varies from 1 minute to 1 minute and 45 seconds.

To Make M/10 Sodium Oxalate: Dissolve 13.4 gm. sodium oxalate in 1 litre of distilled water.

To Make M/40 Sodium Oxalate: Prepare a stock M/10 solution by dissolving 11.1 gm. of anhydrous calcium chloride in 1 litre of water. Then take 25 c.c. of stock solution and dilute to 100 c.c.

Technique: Blood must be drawn quickly and with the minimum of tissue trauma as a small amount of tissue juice in the specimen will spoil the test. The blood must be mixed quickly and thoroughly with the oxalate, as the calcium must be removed immediately, otherwise thrombin forms and remains in the plasma and shortens the clotting time when the plasma is recalcified.

This test is a measure of the concentration of prothrombin and thromboplastin in the blood.

Plasma Index Explanation. We consider that 1.45 is the normal plasma clotting time. Using this time in seconds as the numerator and the patient's plasma clotting time in seconds as the denominator we obtain an index which we call the plasma clotting index. This index normally ranges from

0.8 to 1.05. Readings below 0.8 are on the bleeding side, and those above 1.05 are on the clotting side.

Besides this plasma clotting test, as we call it, we test for fibrinogen and do a platelet count and a platelet lysis determination. We are of the impression that a better understanding of the thromboplastin content of platelets will be a great step in advance in our knowledge.

The test for fibrinogen is as follows:

1. Mix 1 c.c. of plasma with 25 c.c. of 0.9 sodium chloride solution. Add 2 c.c. of 0.25 per cent calcium chloride. Remove clot by wrapping it around a small stirring-rod. Allow it to stand one hour. Remove stirring-rod covered with fibrin. Wash with diluted water.

2. Place stirring-rod in a small tube, add 0.5 c.c. of 10 per cent sodium hydroxide and place in boiling water for thirty minutes. Cool; and add 7.5 c.c. of distilled water.

3. Standard 2 c.c. of standard tyrosin (containing 250 mg. per 100 c.c. of N/10 hydrochloric acid) is mixed with 5.5 c.c. distilled water and 0.5 c.c. 10 per cent sodium hydroxide.

4. To unknown and to standard, add 1 c.c. of phenol reagent[2] and 3 c.c. of 20 per cent sodium hydroxide. Mix and read in colorimeter after thirty minutes.

5. Calculation. $10/R \times 100 \times 0.5$ mg. $\times$ 13: mg. fibrin in 100 c.c. of plasma.

This procedure is a modification of the method of Andersch and Gibson.[3]

Range of fibrin 350 to 750 mg. per 100 c.c.

In order to bring this theoretical consideration of the factors concerned in intravascular blood clotting to a clinical basis we propose to:

(1) Describe our conception of normal and abnormal blood pictures.

(2) Describe our routine examination of patients.

[2] Folin and Ciocalteu. *J. Biol. Chem.* 73: 627, 1929.

[3] *J. Lab. & Clin. Med.*, 18: 816 (May) 1933.

(3) Present a summary of the operative results since we have engaged in this study.

(4) Suggest a therapeutic program for the treatment of thrombosis.

1. The normal plasma clotting index varies from 0.8 to 1.05. Those below 0.8 have a tendency to bleed and those above 1.05 we have considered as potential clotters.

Hemorrhagic Diathesis of Obstructive Jaundice. We have had the opportunity, through the courtesy of Doctor Lewisohn, of Mt. Sinai Hospital, of studying a number of patients besides our own with obstructive jaundice. In attempting to determine the cause of the decreased coagulability of the blood in these cases, the prothrombin of the plasma was quantitatively determined by a special technique. In many of these cases the prothrombin is very greatly diminished, due, we believe, to the damaged liver, which fails to produce a sufficient amount of it to meet the demands of the organism. In other cases of jaundice due to biliary obstruction there is very little if any decrease of prothrombin. The cases where fatality resulted from post-operative bleeding all have diminished prothrombin.

Thrombosis and Embolism. In our previous paper, with the old clotting index, we divided the patients' examination into three types of cases: (a) Patients with normal postoperative indices and having a normal postoperative convalescence. This group comprised 65 per cent of the total routine cases. (b) Patients with high postoperative indices without obvious peripheral blood vessel lesions, approximating 34 per cent. In reviewing the patients with high postoperative blood indices almost universally the convalescence had been abnormal in that there had been a longer continuation of fever than one would expect and the patient had not clinically progressed as one might wish. The patient would often run a temperature of 99.5° to 101°F. from eight to ten days after operation. (c) Patients who had developed thrombosis, thrombophlebitis or embolism. We have had the opportunity of examining 35 patients from our own and other hospitals who have already

developed thrombosis or embolism. All but 2 showed high indices. Eight whose bloods were examined before the onset of the lesion also showed high readings.

2. *Routine Examination of Patients.* It is obvious that before an operation it is impossible to predict what patient is susceptible to thrombosis. The general configuration of the patient and the operation involved may lead one to assume the patient's susceptibility. Nevertheless it seemed necessary in a study of this kind to examine as many patients as possible preoperatively and five and nine days postoperatively. In addition we have had the opportunity of studying many who have already developed thrombosis and embolism, because many of the hospitals in New York, knowing of our interest in this subject, have been good enough to allow us to use their clinical material.

3. *Summary of Cases.* This study was started the latter part of 1928 and has been carried on through 1934. During this period of time 6933 operations have been observed. In this series we have had 3 patients die whose deaths might be attributed to embolism. They are as follows:

CASE I. A man of fifty-eight was operated upon for a left inguinal hernia. While in the hospital his clotting factors were high. Before his discharge his plasma clotting index was 1.12. This was not noted. He returned for the repair of his right side in three weeks and died suddenly on his fifteenth postoperative day. Autopsy revealed pulmonary embolism with infarction.

CASE II. A woman of forty, on whom a cholecystectomy was performed, died on her tenth postoperative day. She had had a moderately high index. She died suddenly, with symptoms strongly suggesting a coronary occlusion.

CASE III. Case treated under the surgical division, but not according to regime adopted as prophylactic for thrombosis. A supravaginal hysterectomy with bilateral salpingo-oophorectomy was performed in December, 1933. Patient had a postoperative ileus and was quite markedly distended. Jejunostomy was considered but was not done because the patient evacuated a moderate amount of gas. On the day before her operation her index was high. She died on her seventh postoperative day, having run a high fever since operation. Autopsy revealed massive pulmonary embolism.

In May, 1934, we started the revised technique of blood examinations: Since that time we have examined 382 cases. There have been 31 cases with high plasma clotting indices, and out of these 31 cases we have had four accidents, viz: First case: One small embolus, followed by normal convalescence. Second case: Patient had three emboli over the period of a month, associated with phlebitis. This patient had a high clotting index during this period. His disease was uninfluenced by sodium thiosulphate. His blood returned to normal after the subsidence of his phlebitis. There were 2 cases of phlebitis unassociated with emboli. Under this new test, then, about 9 per cent of the postoperative cases show high clotting indices and out of this 9 per cent four, or 10 per cent of the highs, have resulted in obvious thrombosis or embolism. These 4 cases occurred almost as an epidemic during the hot months of the last summer. Dehydration was probably an important factor.

4. *Therapeutic Program.* (a) *Prophylactic.* We wish to emphasize the following suggestions for the management of postoperative patients:

(1) In abdominal cases every effort should be made to reduce the postoperative nausea and vomiting, in order to keep the abdominal wall and field of operation quiet. In peritonitis and in the high upper abdominal cases the Levin tube inserted through the nostril immediately after the patient has regained his consciousness, greatly reduces vomiting.

(2) The approach for an abdominal operation is usually through a layer of subcutaneous fat; care should be taken, therefore, to avoid traumatizing the fat by the overzealous pull of the retractors.

(3) The prevention of slowing of the blood stream. Pool in 1913 published an article on "Systematic Exercises in Postoperative Treatment," in which he illustrated the type of exercises to be used, and recommended that treatment be started on the third day after operation. The motion of the

arms and legs would in no way interfere with the healing of the wound and would tend to improve the circulation.

(4) The prevention of venous stasis. In our opinion tight abdominal dressings should be eliminated. With the advent of the Gatch bed, with the patient in a semi-erect position, tight abdominal dressings followed by moderate distention tend to compress the femoral vein as it passes beneath Poupart's ligament. This must of itself create considerable venous stasis. It is our custom to apply sufficient gauze to cover the incision and to hold it in place with enough adhesive plaster to prevent its moving. No attempt is made to apply pressure and no abdominal binders are used. During the five years this procedure has been followed there have been only 4 cases of wound eventration, which were due we believe to other causes. The patients are infinitely more comfortable and their upper abdominal distention is certainly less when pressure is not applied. On the first day postoperative all dressings are inspected and any that feel tight are loosened. Even with dressings applied loosely at the time of operation one is surprised to see an expansion of at least an inch after the adhesive is cut, on the first postoperative day. We believe that distention is lessened if food is given early. Theoretically it is logical to assume that if no food is present in the intestinal tract there is no stimulus for peristalsis, and fermentation will take place. Alvarez and others have shown that when a bolus of food passes through the pylorus, intestinal peristalsis is stimulated, which will carry with it gas as well as solid material. In uncomplicated cases, after spinal, ethylene, or gas anesthesia, supplemented by avertin or pronostin, the patient is routinely given tea and toast the afternoon following the morning operation.

(5) Dehydration. Even in an uncomplicated case the excess of fluid loss from the body over the fluid intake in the first forty-eight hours postoperative is tremendous. If vomiting persists and the temperature is high dehydration very rapidly occurs. It is unnecessary to emphasize the intravenous or

subcutaneous administration of fluid: This is admitted in all surgical clinics. We do advise the early administration of fluid by mouth, if the patient is not vomiting. Some authors have suggested that the intravenous administration of glucose might be one cause of the increased incidence of thrombosis: Experimentally we have been unable to find any increased clotting factors after glucose administration.

When the plasma clotting index is shown to be high, either preoperatively or postoperatively, the patients are placed on a carbohydrate diet, limiting their fats and proteins. It has been shown in dogs and in humans that this diet, which for the sake of convenience we have called a bleeding diet, will diminish the plasma clotting factors while a diet rich in nucleoproteins will cause a marked increase. At the suggestion of Doctor Charles Lieb, of Columbia University, we started using sodium thiosulphate intravenously in the postoperative care of patients with high clotting tendencies. Dr. Lieb had used this drug to prevent extracorporeal clotting in animal experiments and assured us that it was non-toxic. We started to use it more or less empirically. A possible explanation of its therapeutic value is given by Dr. Quick, as follows:

There is an oxidation enzyme called glutathione found in tissues which aids tissue oxidation and prevents the over-production of lactic acid. When this enzyme is depressed there is diminished oxidation and a tendency to decreased capillary permeability, and therefore depressed circulation. Apparently certain toxins and metallic poisons destroy this substance.

Glutathione can, it appears, be influenced by ingested sulphur compounds. Handovsky has increased the liver content of glutathione by various sulphur compounds.

These experiments indicate that sodium thiosulphate can furnish to the body available sulphur, which either neutralizes toxic substances, thus saving glutathione, or contributes sulphur for the synthesis of the latter compound.

Clinically we have used 10 c.c. of a 10 per cent solution of sodium thiosulphate intravenously for three successive days.

repeating the series after an interval of one to two days if results were not satisfactory. We have seen no ill effects from its administration. It is difficult to evaluate the therapeutic benefit of diet and medication in a patient running a slight elevation of temperature, with high clotting factors. Nevertheless, no case where this therapy has been carried on has developed thrombosis or embolism. Whether any of them would have developed this complication if no therapy had been advised is problematical. Clinically the patients have shown improvement from subjective symptoms and by a return to normal temperature in a large percentage of cases within one or two days after the establishment of this regime. The following three brief histories are illustrative of types of patients with high clotting factors who, without the administration of this regime, developed complications:

CASE IV. The first of this group of cases was a man fifty-three years old who was operated on for a right oblique inguinal hernia. A Bassini repair was done, and the patient followed a normal course up until the sixth postoperative day at which time he showed a slight elevation of temperature to 100°. The plasma clotting index on this day was 1.15. The report was sent down late in the afternoon and therefore no therapy was ordered. That night he complained of a sudden attack of pain in the lower chest which was exaggerated on breathing. He had localized dullness with râles. The patient was immediately put on a bleeding diet and given three injections of sodium thiosulphate. His temperature was normal three days after onset, the pain in his chest subsided and he made an uneventful recovery.

CASE V. A young woman of twenty-two was operated on for pericecal adhesions. It was known she had a clotting index of 1.15 on two successive examinations but it was felt she was convalescing favorably and so prophylactic therapy was omitted. She was allowed out of bed on her eleventh postoperative day. On returning to bed she complained of pain in her right thigh and leg, and clinically had a femoral phlebitis. Her temperature went up to 100° and remained there for several days. Sodium thiosulphate was given intravenously and the patient put on a bleeding diet. Her symptoms rapidly subsided and the remainder of her convalescence was uneventful.

CASE VI. The third case was a woman of sixty-five who was operated on for the repair of a cystocele and rectocele. The rectocele wound broke down and there was considerable sloughing and discharge from it, but she

was allowed to go home from the hospital on the eleventh postoperative day. One week after leaving the hospital she developed a phlebitis in her left leg and, just as she was getting up from this, developed a similar condition in her right leg. On reviewing her chart it was found that during the postoperative course her plasma clotting index was 1.15. This had been overlooked, and no prophylactic treatment has been given her.

(b) *Treatment after Complications Arise.* In order to discuss the treatment of phlebitis, thrombophlebitis and embolism it is necessary to concentrate our attention on two clinical groups:

1. Phlebitis and periphlebitis. This type, as generally seen after operation, is characterized by a sharp elevation of temperature, which may persist for five to fifteen days: This rise in temperature is often inaugurated by a chill, and is associated with pain and swelling of the extremities. It is probable that the associated periphlebitis and lymphangitis are largely responsible for the swelling of the limb. Homans has shown that in trying to reproduce this condition experimentally, ligation of the femoral vein does not cause the swelling, but if the adjacent lymphatics in the region of Poupart's ligament are obstructed the cold, swollen leg results which we are accustomed to associated with phlebitis. As this disease is noted early in its onset and as appropriate measures are instituted to put the limb at rest, embolism is not a frequent complication.

2. Thrombosis. It is this type, in which the thrombosis is the predominating factor and the phlebitis a secondary one, that is frequently unrecognized clinically and often results in sudden death from pulmonary embolism. It is in reference to this particular type that we have devoted our biochemical studies.

In these it is felt that diet and the intravenous administration of sodium thiosulphate have been of distinct benefit. It must be understood that elevation of the limb, morphine and cold applied externally are necessary adjuvants of therapy. Moreover, if one vein solely is affected by phlebitis and

proximal ligation with excision can be carried out it is generally accepted that this is a procedure *par excellence*.

We believe sodium thiosulphate will shorten convalescence in cases of phlebitis. When it has been administered immediately after the onset of phlebitis there has been a definite improvement within two days in a considerable percentage of cases. This improvement has been evidenced by relief from pain, subsidence of elevation of temperature and diminution in the circumference of the leg from one to two inches. When it has been administered some time after the onset of the phlebitis, very little benefit has been noted. In cases of embolism we have not noted any great improvement in the majority of cases. We believe that its therapeutic value is largely prophylactic and not curative.

CONCLUSIONS

1. Trauma, venous stasis, dehydration and infection are important factors in the production of thrombosis and embolism. In addition there is a biochemical change in an individual's blood which may render him more susceptible.

2. The plasma clotting test and an analysis of fibrinogen will indicate the susceptible patient.

3. A prophylactic regime is presented which we believe will diminish the incidence of these postoperative calamities.

4. Diet and the intravenous injection of sodium thiosulphate immediately after the onset of phlebitis will shorten convalescence.

The animal experiments in this work were performed in the laboratory of the department of surgery of College of Physicians and Surgeons, Columbia University, New York. We wish to express our gratitude to Dr. Arthur Purdy Stout, Director of the Department.

DISCUSSION

DR. MARGARET STANLEY-BROWN, New York, N. Y.: When this work was started in 1928 we were confronted with such a myriad of problems that it was difficult to know where to begin. From the laboratory side, there was the whole question of just how blood clotted and research in this

field required a more complete knowledge of colloidal chemistry than I possessed. We had a technician who carried on our routine blood test but progress here was slow when you consider that sometimes a month would go by without finding any abnormal indices among our postoperative cases. Through the courtesy of Dr. Stout I was able to do some experimental work with dogs, which I hoped would show what changes in clotting the blood would undergo under different conditions. The work of C. A. Mills had suggested that diet had a marked influence on clotting, so I started out by putting dogs on different types of diets. Before I show the results I want to state that dogs' blood runs a fairly regular parallel to human blood, although the clotting index is higher to start with.

TABLE 1

TESTS MADE ON BLOOD AFTER (1) NORMAL DIET, (2) AFTER FORTY-EIGHT HOURS ON A CARBOHYDRATE AND VEGETABLE DIET, (3) AFTER FORTY-EIGHT HOURS ON A VISCERAL DIET

Dog		Prothrombin	Fibrinogen (Per Cent)	Antithrombin	Platelets	Index
No. 9949	Normal diet	1.11	0.64	1.16	200,000	0.6
	Carbohydrate and vegetable diet	1.11	0.64	1.16	200,000	0.6
	Protein diet	1.45	1.12	0.95	250,000	1.7
No. 9923	Normal diet	1.00	0.54	1.00	400,000	0.5
	Carbohydrate and vegetable diet	0.74	0.54	1.16	220,000	0.3
	Protein diet	1.00	0.64	0.95	300,000	0.7
No. 9924	Normal diet	1.11	0.64	1.16	220,000	0.6
	Carbohydrate and vegetable diet	1.11	0.28	1.00	135,000	0.3
	Protein diet	1.26	0.69	1.00	210,000	0.9
No. 9888	Normal diet	1.00	0.40	1.16	200,000	0.3
	Carbohydrate and vegetable diet	1.00	0.40	1.16	325,000	0.3
	Protein diet	1.11	0.64	0.95	330,000	0.7

The statistics in Table 1 are probably not as accurate as our present ones, for in the recent work Dr. Quick has found that there were many sources of error in doing the plasma clotting test.

Next we took up the question of anesthesia. We were limited in this work to ether because it was the only anesthetic we could get in the labora-

TABLE II

TESTS MADE ON BLOOD BEFORE AND AFTER ONE-HALF-HOUR ETHER ANESTHESIA

Dog		Prothrombin	Fibrinogen (Per Cent)	Antithrombin	Platelets	Platelet Lysis (Per Cent)	Index
No. 9949	before.....	1.11	0.64	1.16	200,000	..	0.6
	after.....	1.26	0.84	1.16	270,000	..	0.9
No. 9923	before.....	1.00	0.64	1.00	380,000	49	0.5
	after.....	1.00	0.60	0.95	505,000	54	0.6
No. 9924	before.....	1.00	0.48	1.16	300,000	48	0.4
	after.....	1.11	0.54	1.00	350,000	60	0.6
No. 9988	before.....	1.00	0.40	1.16	200,000	40	0.3
	after.....	1.00	0.48	1.16	200,000	42	0.4

TABLE III

TESTS MADE ON BLOOD BEFORE AND AFTER TYING OFF APPENDIX AND BEFORE AND AFTER REMOVING SAME APPENDIX AT END OF TWENTY-FOUR HOURS

Dog		Prothrombin	Fibrinogen (Per Cent)	Antithrombin	Platelets	Platelet Lysis (Per Cent)	Index
No. 9996 1st operation	before.....	1.26	0.84	1.00	120,000	25	1.0
	after.....	1.26	0.84	1.00	140,000	29	1.0
No. 9996 2nd operation	before.....	1.45	0.94	0.96	250,000	60	1.4
	after.....	1.45	0.94	0.95	255,000	62	1.4
No. 9991 1st operation	before.....	1.00	0.54	1.00	140,000	36	0.5
	after.....	1.00	0.64	1.00	150,000	40	0.6
No. 9991 2nd operation	before.....	1.11	1.03	0.95	270,000	44	1.2
	after.....	1.11	1.03	0.95	260,000	46	1.2
No. 9979 1st operation	before.....	1.26	1.03	0.95	160,000	39	1.3
	after.....	1.26	1.03	0.95	200,000	50	1.3
No. 9979 2nd operation	before.....		dog died within twenty-four hours.				
	after.....						

tory, but almost always within half an hour the dogs showed a rise in the index.

TABLE IV

TESTS MADE ON BLOOD BEFORE AND AFTER TYING OFF TIPS OF GALL-BLADDER AND BEFORE AND AFTER REMOVING SAME GALL-BLADDER AT END OF TWENTY-FOUR HOURS

Dog		Pro-thrombin	Fibrinogen (Per Cent)	Anti-thrombin	Platelets	Platelet Lysis (Per Cent)	Index
1st operation No. 9923	before	1.26	0.48	1.00	235,000	49	0.6
	after	1.45	0.48	1.00	505,000	58	0.7
2nd operation	before	1.45	0.60	0.91	270,000	60	0.9
	after	1.26	0.60	0.95	370,000	59	0.8
1st operation No. 9888	before	1.11	0.54	1.16	235,000	..	0.5
	after	1.45	0.54	0.95	220,000	..	0.8
2nd operation	before	1.45	0.79	0.87	185,000	..	1.3
	after	2.33	0.84	0.87	275,000	51	2.3
1st operation No. 9924	before	1.26	0.69	1.16	335,000	40	0.8
	after	1.26	0.69	1.16	310,000	37	0.8
2nd operation	before	1.26	0.64	1.00	425,000	41	0.8
	after	1.26	0.64	1.00	450,000	40	0.8
1st operation No. 9991	before	1.11	0.60	1.00	165,000	39	0.6
	after	1.11	0.64	1.00	175,000	43	0.7
2nd operation	before	1.11	0.79	0.95	195,000	38	0.9
	after	1.11	0.79	0.95	250,000	44	0.9

We then did a series of appendectomies and cholecystectomies on dogs and there was a slight rise in the index. However, in both these instances we were working in normal tissues and we wanted to see what the result would be if we worked in pathological tissue, so we tied off a portion of the appendix and a portion of the gall bladder and reoperated in forty-

eight hours on what was then a gangrenous organ. We then got a marked rise in the clotting index.

We then made emulsions of different types of tissues, namely: muscle, subcutaneous tissue and fat; and injected these intravenously into the dogs. The only tissue that gave a change in the clotting was the fat emulsion, and this produced an increase in the tendency to clot. This suggested that possibly the traumatized fat that is reabsorbed after operation affects the clotting index.

Our experience with thromboses was not very successful for it is difficult to produce thromboses in a dog and we never could produce anything which we could honestly call a thrombosis.

Then we took up the study of sodium thiosulphate. We were a little reluctant to start on it without knowing what thiosulphate would do in dogs, so we gave several dogs repeated doses. We gave 5 c.c. of sodium thiosulphate intravenously and took the clotting time again, and there was a rise to 0.5.

We then took up glucose and found that by giving relatively small doses of glucose we got no change at all in the index. Then we wanted to see what the tolerance of the dogs would be to glucose, so we increased the dose to 5 c.c. and repeated it, and then the index would come down to normal but we could never make the dog bleed. We feel relatively safe in using it and are sure it is better than the citrate solutions. (Table v.)

TABLE V

EFFECT OF INTRAVENOUS SODIUM THIOSULPHATE ON THE BLOOD CLOTTING FUNCTION
(Dog No. 10334)

Initial Clotting Index	Experimental Procedure	Final Clotting Index
0.9	5 c.c. $Na_2S_2O_3$ intravenous	0.5
0.67	5 c.c. $Na_2S_2O_3$ intravenous	0.45
0.9	10 c.c. $Na_2S_2O_3$ intravenous	0.7
0.8	150 c.c. 5% glucose intravenous	0.8
0.8	200 c.c. 5% glucose intravenous	0.8
0.9	20 c.c. $Na_2S_2O_3$ intravenous	0.6
0.8	20 c.c. $Na_2S_2O_3$ intravenous	0.6
0.8	20 c.c. $Na_2S_2O_3$ intravenous	0.55

DR. GEORGE A. HENDON, Louisville, Ky.: I wish to make a few remarks relative to thiosulphate phase of this problem. Inspired by the work and the writings of Dr. Wilder Bancroft I began some years ago to use intravenous injections of thiosulphate for the relief of postoperative psychoses.

I was more particularly led to adopt this measure on account of the abundance of experience I was able to obtain in fractures of the hips in old people, and I discovered very soon that it was the best means of controlling the senile psychoses that are so frequently manifested by old people following an injury, either by accident or design. On account of the surprising results I obtained I extended it to include all types of psychoses in connection with infections of various kinds, and in these it seemed to work equally well.

Our methods of administration have differed slightly. We use a continuous intravenous injection of glucose solution, injecting the thiosulphate solution in the Murphy drip, with a hypodermic needle. Just recently we used it in a basal fracture case, giving the patient per venoclysis 7½ grains every hour for seventy-two hours, and it is the only case of basal fracture with hemorrhage that I have ever seen that did not sound the leaden depths of coma or scale the dizzy altitudes of delirium.

Shortly before leaving home I was called upon to take care of a case of hemolytic streptococcic infection involving the entire lower leg from the tubercle of the tibia down to the maleoli. The infection had descended beneath the skin and traveled between the muscular planes. We gave the sodium thiosulphate by intravenous injections one gram every four hours. This morning I received a telegram from my son reporting that the patient is convalescent.

We are not in a position to enter into the detailed experiments that Dr. Bancroft has so well presented, but we did try on one occasion to kill a dog with thiosulphate. This dog weighed 20 pounds and we gave him frequent doses of 75 grains intravenously at a dose, and the only thing it did was to make him wag his tail a little more energetically.

Our experience with this remedy extends over a little more than five years and we have sometimes been startled by its apparent bactericidal effect in the blood stream.

Dr. Roy D. McClure, Detroit, Mich.: We all know the classic work Dr. William Welch did in trying to produce thrombosis and embolism. It seems after his work, that trauma and infection do not play lone rôles in thromboses in man.

I feel that today we have several practical means of prevention that are often overlooked. Dr. Bancroft spoke of dehydration. I think it is a common error to have patients returned from the operating room to a bed of hot water bottles and too many hot blankets. That patient unless carefully watched perspires too freely, contributing to the dehydration. This can be prevented by blanket regulation and by the use of intravenous fluids.

I worked for several years with Dr. Pool in New York Hospital, and was greatly interested in his prescribing exercise for his patients. Dr. Bancroft said he believed in these exercises. Our patients have massage of the body, arms and legs immediately on return from the operating room. Private patients have regularly professional massage.

Dr. H. A. Gamble, Greenville, Miss.: There is nothing more tragic than to see a patient practically able to return to his home after undergoing some operative procedure, usually abdominal, fall back in bed gasping for breath and in a short while expire.

We are cognizant of the fact that there are a number of factors involved in the causation of these complications—among which are the changes in the blood serum, changes in the constituent elements of the blood, changes in those elements which go toward favoring clotting postoperatively and probably most important of all the retardation of the blood stream.

We have approached the problem from a different angle from that of Dr. Bancroft. We plan definitely from the outset of any operation to employ consecutively all measures which we feel will have a preventive effect upon the occurrence of thrombophlebitis and pulmonary embolism. First, in all operations there is a large loss of body fluids with a lowering of the water level of the system and a concurrent increase in the viscosity of the blood. In order to prevent this as far as is possible a 5 per cent solution of glucose is given subcutaneously throughout all operations of a major character (the amount varies as to the rate of absorption from 1000 c.c. to 2000 c.c. per patient). During the operation we feel that all manipulations, application of forceps, etc., should be attended by as little trauma as possible as it is largely here that thrombokinase originates, is absorbed, and increases the coagulability of the blood.

Postoperatively, in order to prevent the retardation of the flow of blood which in our opinion is one of the most important factors in the production of thrombi, and knowing that the flow of venous blood to the heart is largely dependent upon the negative pressure of respiration and the maintenance of the muscle tone and contraction of the muscles of the lower limbs, we first, systematically, give inhalations of carbon dioxide and oxygen for five minutes every two hours for the first twenty-four hours. Originally this was instituted as a preventive of pulmonary complications. Whether it is of any service for that purpose or not is a mooted question but there is no doubt that it materially speeds up the venous flow of blood.

Second, after the first twenty-four hours we require all patients twice daily to systematically exercise their lower limbs upon a mechanical device consisting of two bicycle pedals mounted upon a broad base. The use of massage as mentioned by Dr. McClure would no doubt be equally effective. This, however, is satisfactory, does not require extra nursing

force, and besides the patients all like it and are in better physical condition when allowed up than would otherwise be the case.

By these methods:

1. Maintaining of body fluid level at time of operation by administering fluids subcutaneously.

2. Setting free as little thombokinase as possible by avoidance of unnecessary trauma.

3. Promoting the flow of venous blood by the forcing of deep respiration with carbon dioxide and oxygen administered systematically.

4. The prevention of venous stasis in the lower limbs by systematic pedalling exercises; we believe that we have practically eliminated these postoperative complications, thrombophlebitis and pulmonary embolism.

Dr. Frederic Bancroft (*Closing*): I was asked if we had any patients with a normal index who showed any evidence of phlebitis. One patient with a normal index but with a high platelet count and platelet disintegration rate had a phlebitis. This was not on our service but was a case we saw in consultation. An amputation of the breast, started with a chill and high temperature. We felt this was largely due to infection and not so much due to thrombosis.

I was interested in what Dr. Hendon said relative to the use of sodium thiosulphate in cases of mental excitation following fracture. Dr. Murray and Dr. Darrach, on the fracture service of the Presbyterian Hospital, had utilized our technique for blood examinations. In many cases of fracture they found symptoms that might suggest either delirium tremens or psychosis, the clotting factors were universally high and relief was obtained by the intravenous use of sodium thiosulphate and the institution of a bleeding diet. We should like to emphasize that we do not wish to put too much stress on the clotting factors. In prophylactic treatment one must attempt to prevent venous stasis, dehydration and infection. We believe, however, that the plasma clotting index is as accurate a means of determing a patient's clotting tendencies as the leucocyte count is an indication of infection.

SCALENUS ANTICUS (NAFFZIGER) SYNDROME*

ALTON OCHSNER, M.D., F.A.C.S., MIMS GAGE, M.D., F.A.C.S. AND MICHAEL DEBAKEY, M.D.

NEW ORLEANS, LA.

THE presence of supernumerary ribs in the cervical region has been known since the original observations of Willshire[1] to cause symptoms in some cases because of the pressure exerted by the rib on the brachial plexus and subclavian artery. Clinicians have, however, repeatedly observed that cervical ribs may occur without producing symptoms and that in many cases with a unilateral cervical rib symptoms may occur on the contralateral side in which no rib is demonstrable. Torelli[2] reported 100 cases of cervical rib, only 9 of which produced symptoms. Adson and Coffey[3] found that cervical rib occurs more often on the left side, but symptoms are present more commonly on the right side. The fact that pressure symptoms identical with those produced by cervical ribs can occur in the absence of supernumerary ribs in the cervical region had led many[4–14] to conclude that the subclavian structures are compressed by the first rib. The position of the first thoracic rib is only relative and even though it may be elevated with respect to the vertebral column, few, if any, symptoms will result unless it is raised with respect to the subclavian structures as symptoms occur only when the brachial plexus and subclavian artery are compressed. Conversely, in the absence of elevation of the first rib with respect to the vertebral column, symptoms occur if there is depression of the shoulder with a resulting pull on the subclavian structures or a relatively low origin of the brachial plexus, causing angulation over the first rib. Thus it may be concluded that symptoms occur only when there is alteration

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in the relative positions of the subclavian structures and first dorsal rib. Our interest in this condition was stimulated by the observation of a case (Case 1) in which no cervical rib could be demonstrated, but in which a typical cervical rib syndrome was present. Because of the bizarre manifestations which could not be explained the patient was considered neurotic. The true condition was suspected only after an informal discussion with Howard Naffziger[15] of San Francisco, who related the histories of 2 similar personal cases in which complete relief had been obtained by sectioning the scalenus anticus muscle, a procedure which was advocated by Adson and Coffey[3] in 1927 for the relief of cervical rib symptoms. Naffziger[15] believed that the symptoms in his cases were caused by pressure on the brachial plexus and subclavian artery by the scalenus anticus muscle, as Adson and Coffey[3] had previously considered that the symptoms in cases with cervical rib resulted from compression of the subclavian structures in the angle between the scalenus anticus muscle and cervical rib. Carroll[14] reported 2 cases of cervical rib syndrome without cervical ribs, symptoms resulting, according to him, from a high placed first thoracic rib. Scalenotomy was performed in one of these cases in which there was apparently an osteoma of the first rib with complete relief of all symptoms. Since the observation of our original case, we have had 5 additional cases of scalenus anticus syndrome.

Two widely quoted theories based upon anatomical dissections concerning the symptoms resulting from cervical ribs have been described by Todd[16] and Jones,[17] respectively. According to Todd,[16] compression of the subclavian structures results from an abnormal development of the shoulder girdle. Normally during the intra-uterine and pre-adolescent development the acromial end of the clavicle and shoulder descend because of the weight of the upper extremity and the sternal end of the clavicle descends because of the contraction of the rectus abdominus muscle exerted through the sternum. No symptoms occur, unless there is a greater descent of the

shoulder or an arrested descent of the sternum and the anterior ends of the ribs. Either one or both of these abnormalities results in compression of the subclavian structures because of the stretching of the brachial plexus and subclavian vessels over a fixed cervical or first dorsal rib. Todd[16] believes that cervical rib syndrome occurs more frequently in females than in males because the descent of the shoulder is greater in the former due to lack of development of the suspensory muscles of the shoulder, especially the trapezius. The rectus abdominus muscle being relatively poorly developed in the female exerts less pull on the sternum, causing early high fixation of the anterior extremities of the clavicle and ribs. Jones,[17] in extensive phylogenetic, embryologic, and anatomic investigations attempted to explain the development of cervical ribs and symptoms in cases with cervical ribs and those without cervical ribs as resulting from an abnormal development of the brachial plexus. In those cases in which the brachial plexus principally originates from the cervical segments of the spinal cord, no symptoms occur, whereas in those cases in which a considerable portion of the lower cord of the brachial plexus is derived from the upper thoracic segments of the cord, symptoms are apt to result from compression and angulation of these nerves over the first thoracic or cervical ribs. Normally, a slender branch joins the brachial plexus from the first and occasionally the second thoracic nerves. According to Jones,[17] symptoms resulting from compression of the lower cord by cervical or first thoracic ribs occur only when the thoracic branches are abnormally large or the entire root joins the brachial plexus. John B. Murphy,[18] in 1906, considered the vascular manifestations associated with cervical rib the result of pressure of the subclavian artery against "a rigid scalenus anticus muscle" and that the artery and not the vein was involved because the former is posterior and the latter anterior to the muscle. Adson and Coffey,[3] in 1927, explained the symptoms in cases with cervical rib by compression of the subclavian structures in the angle between the scalenus anticus

muscle and the rib. They advocated division of the scalenus anticus muscle as the treatment of choice, which procedure permitted the brachial plexus and subclavian artery to glide

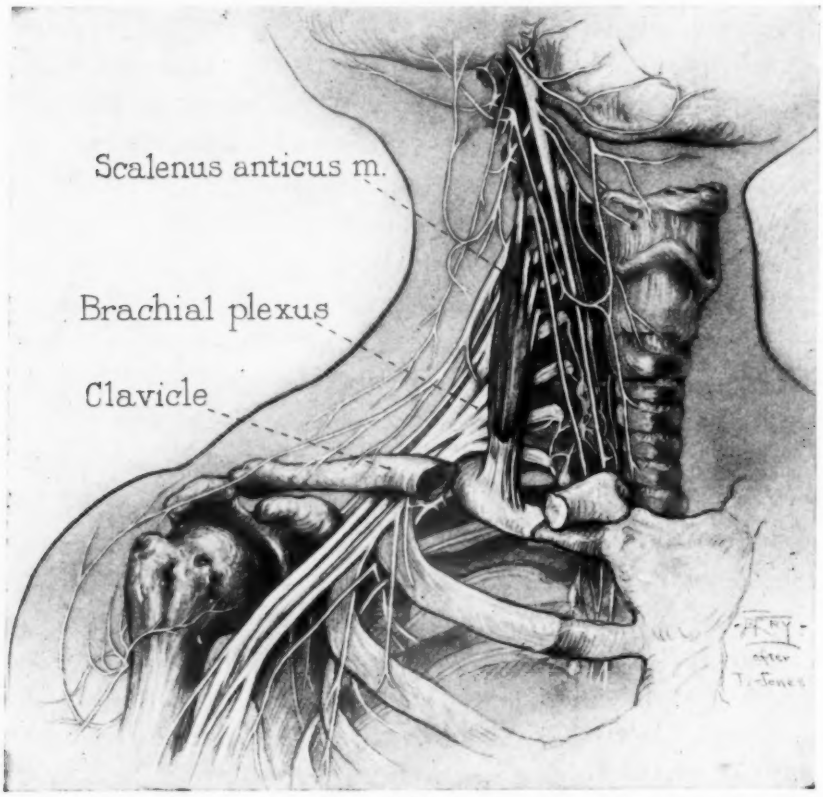

FIG. 1. Angulation of lower cord of brachial plexus over first rib as result of elevation of rib caused by contraction of scalenus anticus muscle. Plexus is also pinched between muscle and first rib. (Modified after Adson and Coffey.)

forward on the superior surface of the rib.

The foregoing theories have been advocated to explain the development of symptoms in patients with cervical rib, but undoubtedly in many cases they also explain the typical syndrome occurring in those patients with no cervical rib. From our own clinical observations, we believe that the afore-mentioned agents are predisposing factors toward the development of symptoms in individuals with no cervical ribs. The fact that symptoms are not, as a rule, manifest until

adulthood suggests an additional exciting factor. This we believe is contraction and spasm of the scalenus anticus muscle, which results from irritation and stimulation of the

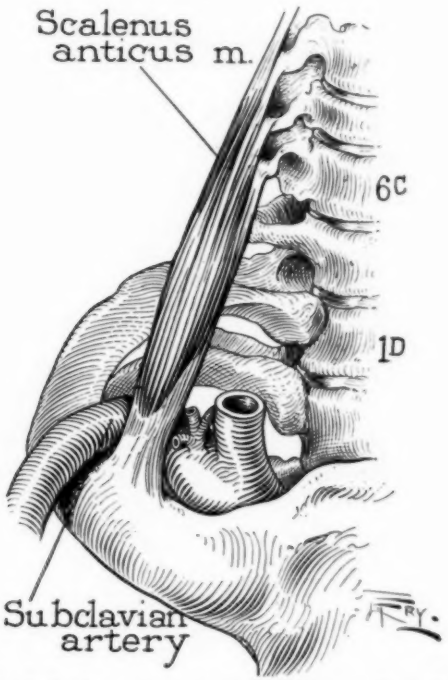

FIG. 2. Compression of subclavian artery in angle between first rib and scalenus anticus muscle resulting from an elevation of first rib by contraction of scalenus anticus muscle. Brachial plexus is omitted in order to show compression of vessel alone.

brachial plexus, some of the fibers of which supply the scaleni muscles. This produces an abnormal elevation of the first dorsal rib which in turn causes greater irritation and stimulation of the brachial plexus, establishing a vicious circle. This contention is based upon the finding in all of our cases of an abnormally well-developed, spastic, and stiffened scalenus anticus muscle and the sudden and marked descent of the first rib following division of the muscle. Because of the prominent rôle played by the scalenus anticus muscle as an active exciting factor in the elevation of the first rib, because of the pressure it exerts on the subclavian structures, and because section of it relieves the symptoms, we believe that

the condition should be termed scalenus anticus syndrome. Spasm of the scalenus anticus and medius muscles will aggravate the pressure symptoms not only because of elevation of the rib but also because the brachial plexus and subclavian artery are pinched between the two muscles. The symptoms in cervical rib and scalenus anticus syndrome are the result of compression of the brachial plexus and subclavian artery (Figs. 1, 2). This compression may be due to stretching of these structures by an abnormally low position of the shoulder, to high fixation of the sternum and ribs (Todd[16]), to low origin of the brachial plexus (Jones[17]), and to spasm of the scaleni muscles, resulting from brachial plexus irritation. Undoubtedly, many if not all of these factors are responsible in the majority of instances.

PATHOLOGIC ANATOMY

The pathologic changes occurring in scalenus anticus syndrome are not well known, because the subclavian artery and brachial plexus have not been removed for study. Angulation and flattening of these structures over the first rib are almost invariably present and produce a traumatic neuritis in the distribution of the lower segments of the brachial plexus. A grooving of the first rib, because of the pressure of the brachial plexus, has been described by Todd[19] and Jones.[20] In all of our cases we have found a thickened, spastic scalenus anticus muscle which appeared of firmer consistency than normal. In one of our cases the examination of the muscle removed at operation showed degeneration and replacement of the muscle fibers by connective tissue (Fig. 3). Atrophy of the muscles of the arm, forearm, and especially the intrinsic muscles of the hand, is occasionally observed. Aneurysm of the subclavian artery, just distal to the compressed area, has been observed as occurring in cases with cervical rib, but as yet has never been reported in patients with scalenus anticus syndrome.

CLINICAL MANIFESTATIONS

Scalenus anticus syndrome occurs more frequently in women than in men. All of our 6 patients were women. The

FIG. 3. Section of scalenus anticus muscle stained by hematoxylin eosin. Low power photomicrograph showing atrophy of muscle bundles with replacement fibrosis indicated by arrows.

lesion is usually on the right side; in our cases the right side was involved 4 times and the left side twice. Young adults are most frequently affected. In our series the ages varied from seventeen to forty-seven years, the average being 28.8 years. The symptoms and signs in scalenus anticus syndrome consist of two main groups: (1) the nervous and (2) the vascular; and are the result of involvement of the brachial plexus and the subclavian artery, respectively. The nervous manifestations are by far the more consistent and prominent. Pain is the principal symptom and is usually referred to the shoulder, supraclavicular region, down the arm, ulnar and flexor surfaces of forearm to hand, and fre-

quently to the side of the neck and ear. It may vary from an uncomfortable tingling, numb sensation to severe, lancinating pain. In our 6 cases pain was the most prominent symptom. In

FIG. 4. How sweeping will aggravate condition of scalenus anticus syndrome by causing a dropping of right shoulder. This increases drag on brachial plexus and subclavian artery aggravating angulation over first rib and causing an exacerbation in symptoms.

6 the pain was in the arm, in 5 it was in the supraclavicular and shoulder areas, radiating to the forearm and hand in each instance. In 2 cases the pain extended to the neck. The pain was described as intense in 3 and dull in 3. In 4 of the 6 cases the pain varied, being at times dull and again intense. In 2 instances it was necessary for the patient to carry the arm in a sling because of the severe pain. In every instance there

was a marked supraclavicular tenderness on pressure over the scalenus anticus muscle, which resulted in radiation of the pain down the ulnar side of the arm. Atrophy of the deltoid muscle

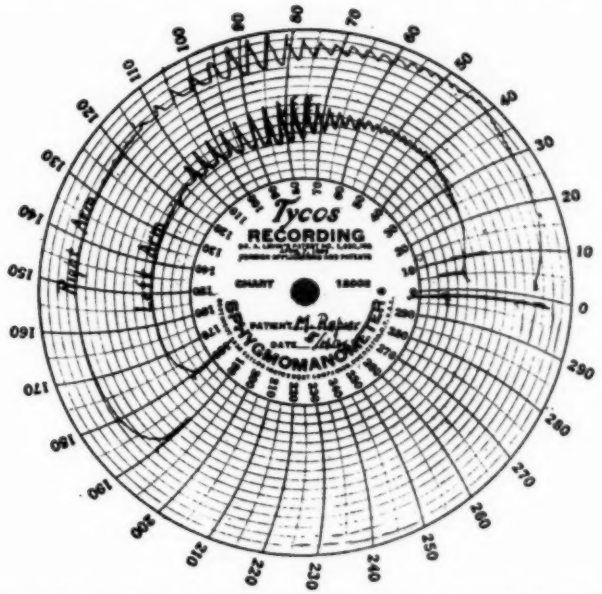

FIG. 5. O. M. G. of right and left brachial arteries before operation. Oscillometric index on right is of smaller amplitude than left, indicating that there is a diminished pulse pressure suggestive of arterial compression by first dorsal rib and scalenus anticus muscle.

was observed in only one instance. The pain was frequently aggravated by turning the head toward the affected side with the neck extended and by deep inspiration. In all of our cases there was an increase in pain when the shoulder was depressed. In 2 of our patients one of the prominent manifestations was an increase in pain upon sweeping (Fig. 4) which disappeared after division of the scalenus anticus muscle. Whereas the nervous symptoms are largely due to pressure on the inferior trunk of the brachial plexus which explains the pain on the ulnar side of the forearm and hand, more extensive involvement of the brachial plexus may result. Compression of the entire brachial plexus results either from transmitted pressure through the subclavian artery or from a squeezing of the brachial plexus between the thickened and stiffened scalenus anticus and

medius muscles. Some of the bizarre nervous manifestations may be due to an overflow of impulses to other portions of the brachial plexus as a result of continuous and excessive stimula-

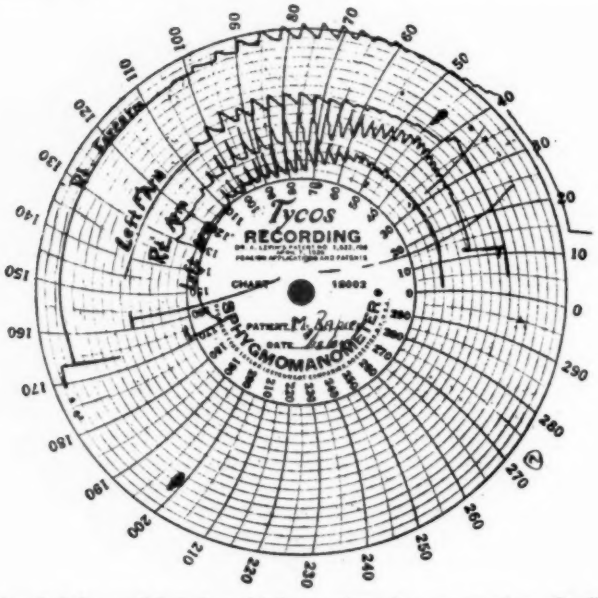

FIG. 6. O. M. G. of right and left brachial arteries after operation. Oscillometric index of right side is of greater amplitude than left, demonstrating that compression present preoperatively has been relieved by scalenotomy.

tion of the lower cord (Head) and also possibly as a result of sympathetic involvement.

Vascular manifestations consist of diminution of the pulse volume on the affected side, decrease in surface temperature, numbness, coldness, and formication. The vascular manifestations are much less prominent than are the nervous symptoms. They are the result either of direct compression of the subclavian artery by the rib and scalenus anticus muscle (Fig. 2) as originally described by John B. Murphy,[18] or the result of sympathetic stimulation (Todd[21]), or a combination of both. Sympathetic stimulation is undoubtedly responsible for many of the vasomotor disturbances. Coldness of the fingers was observed in 4 of our 6 cases; numbness of the extremity in 3; tingling in 2. As already mentioned, the vascular changes are not striking and frequently are overlooked unless special

methods and precautions are taken to elicit them. We have found that the diminution in pulse volume as determined by oscillometrograms (O.M.G.) with the Tycos Recording Sphygmomanometer is the most accurate means of eliciting the early vascular changes. These determinations should be made on both arms and forearms, before and after cervicodorsal sympathetic novocaine block. The latter procedure is done to ascertain the degree of sympathetic vasospastic influence. Characteristically the alteration of the oscillometric index, as determined by O.M.G., consists of general decrease in oscillation but particularly of decreased oscillations at the high blood pressure levels (Figs. 5, 6).

DIAGNOSIS

The diagnosis of scalenus anticus syndrome is not difficult, if the condition is only kept in mind and those conditions with which it is apt to be confused are eliminated. These are: cervical rib, subacromial bursitis, supraspinatus tendon rupture, cervicodorsal sympathalgia, Raynaud's disease, and brachial neuritis. The clinical picture in cases with cervical rib is identical with that seen in scalenus anticus syndrome, the mechanism of their production being the same, the only difference being one of degree in that occasionally cervical ribs produce more marked symptoms. Absence of cervical ribs in cases of scalenus anticus syndrome can be demonstrated by roentgenography. As a matter of fact, a patient presenting a cervical rib syndrome and in whom a cervical rib cannot be demonstrated, probably has a scalenus anticus syndrome. The clinical picture in subacromial bursitis and supraspinatus tendon rupture simulates that of scalenus anticus syndrome in that the pain in both is apt to be referred to the shoulder. In the former, however, tenderness is limited over the supraspinatus tendon and subacromial bursa without other accompanying nervous and vascular manifestations. Atrophy of the deltoid may be a prominent symptom. Abduction of the arm increases the pain in cases with subacromial bursitis, whereas

this position relieves the pain in scalenus anticus syndrome (Fig. 7). In cases with subacromial bursitis, roentgenograms frequently show calcification in the region of the tendon and

FIG. 7. Elevation of shoulder, which, because of diminished tension on brachial plexus and subclavian artery, prevents angulation over first rib and produces relief in symptoms.

bursa. Cervicodorsal sympathalgia must be differentiated from scalenus anticus syndrome because of the nervous and vascular manifestations which are similar in both. In the former condition, pain originating in the supraclavicular region radiating to the neck and face and also down the arm may be encountered, accompanied by vasomotor manifestations. This condition can be easily eliminated by the complete relief of symptoms following novocaine block of the cervicodorsal sym-

pathetic ganglia. Every patient with a scalenus anticus syndrome should be given the advantage of this procedure. This was done in all of our cases which were operated upon. Raynaud's disease, because it also most frequently occurs in women and because of the vasomotor disturbances, may be confused with scalenus anticus syndrome. Raynaud's disease usually is bilateral, whereas the scalenus anticus syndrome is usually unilateral. The nervous manifestations in Raynaud's disease are absent. In all cases of brachial plexus neuritis, the possibility of scalenus anticus syndrome must be considered and eliminated. In addition to general physical examination, *x*-ray, and sympathetic block of the cervicodorsal sympathetics, we feel that careful oscillometric examination of both arms and forearms, before and after sympathetic block, is of great diagnostic importance in cases with scalenus anticus syndrome. The diagnostic vascular changes consisting of diminution in, and at times complete absence of the radial pulse can be produced by rotating the head toward the affected side and extending the chin. Oscillometrograms (O.M.G.) are indispensable for the accurate determination of the vascular changes. In all of our cases in which it was done a definite decrease in the oscillometric index as determined by oscillometrograms was observed. Of great diagnostic importance is the persistent localized point of tenderness over the scalenus anticus muscle in the supraclavicular space, with radiation of the pain into the arm.

PROGNOSIS

The prognosis in scalenus anticus syndrome as regards morbidity is not good unless therapy is instituted. Because of the vicious circle set up, as described, resulting from spasm of the scalenus anticus and medius muscles produced by irritation and stimulation of the brachial plexus, elevation of the first rib with increase in nervous and vascular symptoms results. Unless this vicious circle is broken the condition will

become progressive. Following adequate surgical therapy the prognosis is good as regards mortality and morbidity.

TREATMENT

Brickner and Milch[12] advocated conservative treatment for the relief of symptoms in cases with no cervical rib on the assumption that development of the suspensory muscles of the shoulder girdle favored the elevation of the shoulder to its normal position and relieved the stretching and subsequent compression of the subclavian structures. It is our opinion that this method of therapy offers relatively little and undoubtedly must be prolonged. We feel, because of the prompt and complete relief of pain following the operative therapy, that this method is to be preferred to the conservative ones. We divided the scalenus anticus muscle in 4 of our 6 cases in order to break the vicious circle as described above. This procedure gave gratifying results. Operation has been advised in the remaining 2 cases but as yet has not been performed. Adson and Coffey[3] are of the opinion that the beneficial results obtained in cases of cervical rib by scalene section are produced by the forward displacement of the brachial plexus and subclavian artery over the site of attachment of the scalenus anticus muscle. We, however, are of the opinion that the beneficial results obtained in the cases of scalenus anticus syndrome are due *to the break in the vicious circle which allows the first rib to assume a lower position*, thus relieving the pressure both on the subclavian artery and the brachial plexus. This assumption is based on the operative observation of the sudden depression and disappearance into the depths of the wound of the first rib following division of the scalenus anticus muscle.

The technique of operation as performed in our clinic is as follows: A transverse incision, approximately 6 cm. long, is made in the supraclavicular fossa, approximately 2 cm. above and paralleling the clavicle with its midportion over the posterior border of the sternocleidomastoid muscle (Fig. 8). After incising the skin, subcutaneous fascia, and platysma, the

posterior border of the sternocleidomastoid is exposed and retracted medially. If necessary, which is seldom the case, the posterior half of the sternocleidomastoid may be incised. The

FIG. 8. Skin incision for scalenus anticus resection. Incision is made just above clavicle with its midpoint over posterior border of sternocleidomastoid muscle.

areolar tissue deep to the posterior border of the sternocleidomastoid is palpated and the scalenus anticus muscle is identified at its attachment to the first rib by digital examination in the depth of the wound. The areolar tissue is bluntly dissected, care being taken not to injure the thyrocervical trunk. Frequently it is necessary to ligate the transverse cervical artery. Occasionally it is necessary to retract the omohyoid muscle laterally. It has not been necessary to divide the omohyoid in any of our cases. After exposing the anterior surface of the scalenus anticus muscle, the phrenic nerve is identified crossing its anterior surface from above downward and medially. It is

mobilized and retracted medially (Fig. 9). By means of the finger, the lower end of the scalenus anticus muscle is separated from the subclavian artery and brachial plexus and is carefully

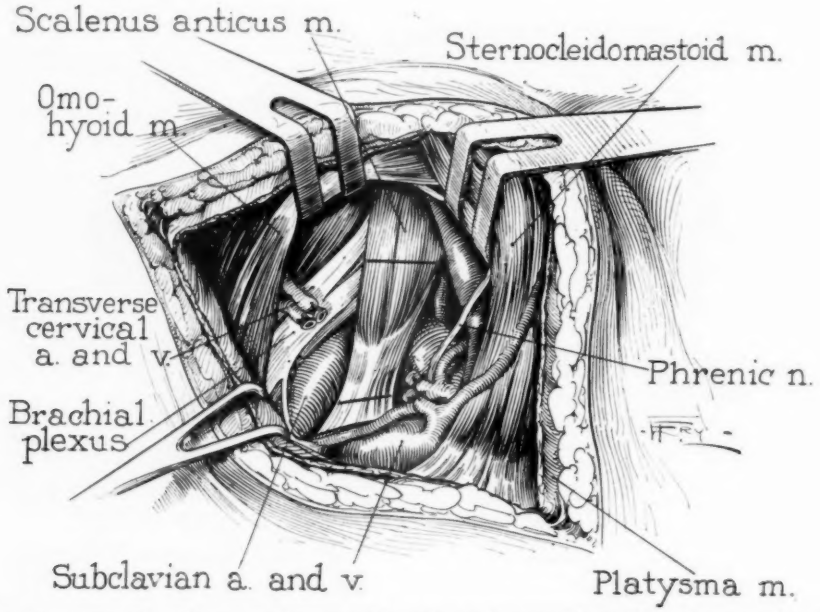

FIG. 9. Exposure of scalenus anticus muscle prior to its resection. Phrenic nerve has been mobilized from anterior surface of muscle and is retracted medially by means of a retractor. Omohyoid muscle is retracted laterally by retractor in upper and left hand corner of wound. Transverse cervical artery and vein have been ligated, exposing scalenus anticus in region of its attachment to first rib. As shown in illustration, subclavian artery and brachial plexus are pinched in angle between thickened scalenus anticus muscle and first rib.

inspected concerning its size, consistency, and tone. (In one of our cases the scalenus anticus was larger than the sternocleidomastoid muscle.) After carefully separating the subclavian artery and brachial plexus from the posterior aspect of the scalenus anticus muscle a curved spatula or retractor is passed around the posterior surface in order to protect the subclavian artery. The muscle is divided close to its insertion on the first rib by cutting a small portion at a time in order to prevent injury to the underlying vessels and nerves. We believe it is desirable not only to divide but also to resect the distal portion of the scalenus anticus muscle (Fig. 10) because

of the possibility of fibrous bridging between the two ends of the divided muscle resulting from organization of exudates; i.e., blood and serum. This is done because there is a possibility

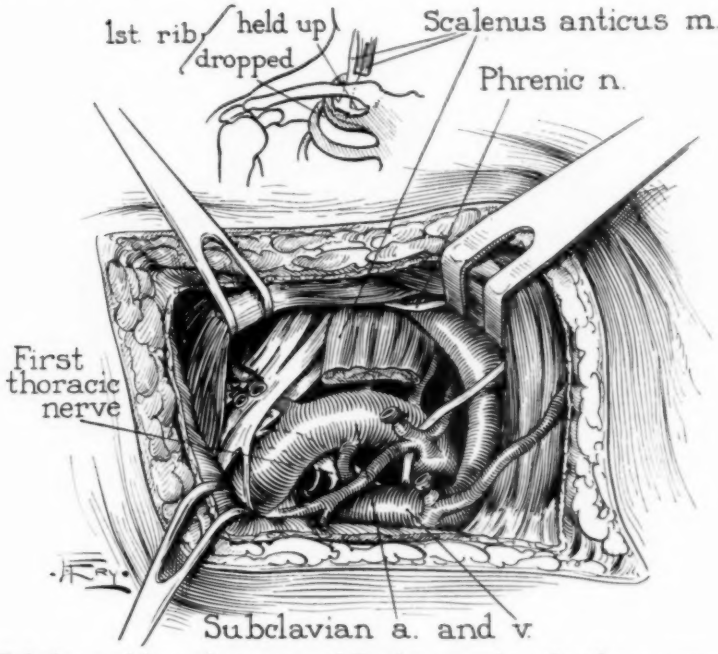

FIG. 10. Relative positions of structures following resection of scalenus anticus muscle. Attachment of scalenus anticus muscle and first rib has disappeared into depths of wound, due to release of tension produced by contraction of muscle. Subclavian artery and brachial plexus resume their normal positions.

of postoperative oozing from the cut muscle resulting in hematoma between the muscle ends. We believe that in one of our cases recurrence of symptoms after several weeks of complete relief of symptoms was due to this fibrous bridging, and feel that had a portion of the muscle been resected the recurrence could have been prevented.* In the operative treatment of torticollis resection of the sternocleidomastoid is practiced in order to prevent a similar condition from occurring. Careful inspection of the brachial plexus should be done to determine the presence of other lesions, especially adhesions, and if the

*The patient has subsequently been operated upon, and at operation a bridging of the muscle defect by fibrous tissue was found. Following extirpation of this and the adjacent ends of the muscle, the patient was completely relieved.

latter are found they should be freed. Careful hemostasis of the wound is secured as it is essential that hematoma formation be prevented in order to minimize the amount of fibrous tissue in the wound. The deep fascia is sutured by means of interrupted oo chromic catgut sutures, the platysma is closed by means of interrupted oo chromic catgut sutures, and the skin is closed by means of Michel clips or subcuticular sutures.

COMPLICATIONS

The complications which may occur, but which can be readily avoided by paying careful attention to operative technique consist of injury to the subclavian vessels, phrenic nerve, internal jugular vein, carotid artery, thyrocervical trunk, and pleura.

SUMMARY AND CONCLUSIONS

1. Scalenus anticus syndrome is a clinical entity, the symptoms of which are identical with those of cervical rib.

2. The symptoms are the result of compression of the brachial plexus and subclavian artery on the first thoracic rib. This compression may be due to abnormally low position of the shoulder, to high fixation of the sternum and ribs, to low origin of the brachial plexus, and to elevation of the first thoracic rib due to spasm of the scaleni muscles, resulting from brachial plexus irritation. The first three conditions are predisposing, whereas the last is an exciting factor.

3. Irritation and stimulation of the brachial plexus by pressure of the first rib causing spasm and shortening of the scalenus anticus muscle results in elevation of the first rib, thus establishing a vicious circle.

4. Vascular changes are frequently present but are usually missed unless oscillometric studies are made.

5. Scalenus anticus syndrome must be differentiated from cervical rib syndrome, subacromial bursitis, supraspinatus tendon rupture, cervicodorsal sympathalgia, Raynaud's disease, and brachial plexus neuritis.

6. The treatment consists of resection of a portion of the scalenus anticus muscle.

7. Six cases of scalenus anticus syndrome, 4 of which have been successfully operated upon, are reported.

CASE 1. L. R., white female, aged twenty years, single, house work. April 20, 1933: Admitted to Hutchinson Memorial Clinic.

Chief Complaint: Pain in right shoulder.

Present Illness: In April, 1932, while playing "pop-the-whip" fell and lost consciousness for ten minutes. When she recovered had burning, sticking pain in right shoulder and could not move shoulder or arm. Admitted to a hospital where she remained four days. Right arm placed in extension splints and after *x*-rays were made patient was told she had contusion of elbow and discharged to outpatient department for "baking" of arm. Pain in right shoulder radiating to arm and neck has persisted to present time.

Physical Examination: Asthenic habitus, well nourished, and of normal stature. Temperature, pulse, and blood pressure normal.

Right arm held close to side, forearm flexed, and patient not inclined to move extremity. No limitation of motion in elbow or wrist, but there is pain at shoulder on active or passive motion. Slight atrophy of deltoid. Tenderness in right supraclavicular fossa. Exquisite tenderness over attachment of the scalenus anticus muscle. Pain radiates to ulnar side of arm and forearm.

X-ray examination negative for cervical rib or osseous lesions.

May 1, 1933: Patient fainted during examination and subsequent events led examiner to believe patient to be psychotic. Patient carries right arm in sling.

June 6, 1933: Patient continued to complain of pain. Right hand definitely colder than left. Persistent tenderness in right supraclavicular fossa.

September 9, 1933: Because of pain in shoulder, subacromial bursitis suspected. Tenderness not marked over bursa, however. Because of bizarre manifestation with radiation to neck cervicodorsal sympathalgia suspected.

October 2, 1933: Tulane Surgical Service, Touro Infirmary. Cervicodorsal sympathetic ganglion block with novocaine. No relief of symptoms.

October 29, 1933: Because the manifestations were typical of cervical rib syndrome but as no cervical rib could be demonstrated roentgenologically, a diagnosis of scalenus anticus (Naffziger's) syndrome was made.

December 19, 1933: (Tulane Surgical Service, Touro Infirmary.) Operation under gas anesthesia. Incision over posterior border of sterno-

cleidomastoid muscle with division of the clavicular portion of this muscle. Section of the scalenus anticus muscle was followed by disappearance of the first rib into the depths of the wound.

December 24, 1933: Discharged from hospital. Convalescence has been uneventful. Completely relieved of all symptoms.

June 25, 1934: Patient continues to be free of all symptoms. Is able to do all house work.

COMMENT: The true condition of this patient was not suspected until after the coversation with Naffziger already mentioned.

CASE II. R. F., white female, aged thirty-three years, married, saleslady.

March 23, 1934: Admitted to the Tulane Surgical Service at Charity Hospital.

Chief Complaint: Pain in right shoulder.

Present Illness: Two years ago while swimming, right arm was "jerked" backward. No pain at that time, but next day could not turn her head to the right because of pain in neck and right shoulder. This lasted about six months. A week ago, woke up with pain in right shoulder, radiating down the right arm to wrist. Pain intense, but the whole area felt numb. pain relieved by putting hand to back of head.

Physical Examination: Temperature and pulse normal on admission. Blood pressure 122/84. Tenderness in right supraclavicular fossa limited to attachment of scalenus anticus muscle.

X-ray shows cervical rib on the left but none on right.

March 29, 1934: Novocaine block of right cervical and upper dorsal sympathetic ganglia, following which right upper extremity became warmer and drier than the left. Typical Horner's syndrome appeared. O.G.M. taken before and after the block shows definite increase in amplitude of arterial oscillations. As patient was having no pain at time of injection the influence of the injection on pain could not be determined.

April 4, 1934: Operation. Under gas anesthesia incision was made in the supraclavicular fossa over the posterior border of the sternocleidomastoid muscle. This was retracted medially. The transverse cervical artery was doubly ligated and divided. The scalenus anticus muscle was freed and divided near its insertion on the first rib, following which the rib disappeared out of sight into the depths of the wound.

April 12, 1934: Postoperative convalescence has been uneventful. Patient discharged from hospital today.

May 5, 1934: Communication from patient. "I am very glad to be able to tell you that the arm and shoulder have not caused any further pain or trouble."

COMMENT: This case is of interest because of the presence of a small cervical rib on the left side although the patient had no symptoms on that side, all the manifestations being on the right, on which side no cervical rib could be demonstrated.

CASE III. R. S., white female, aged forty-seven years, married, house work.

April 9, 1934: Admitted to the Tulane Hutchinson Memorial Clinic.

Chief Complaint: Swelling of left supraclavicular fossa.

Present Illness: About September, 1933, began to notice swelling of left supraclavicular region. Believes swelling slowly became larger. Symptomless until two months ago (February, 1934) when she began noticing dull, gnawing, uncomfortable aching sensation in this area. One month later this pain radiated down left arm. Tingling and numb sensation in left hand and fingers. Coldness of left hand.

Physical Examination: Temperature, pulse, respiration normal.

Palpation in left supraclavicular region reveals definite tenderness over attachment of scalenus anticus muscle with pain radiating down left arm. Left hand is colder than right. Numbness apparently more marked along ulnar distribution of forearm and hand. Pain in the left supraclavicular region and shoulder and arm is relieved upon elevating the left arm. No noticeable differences in sensations. Reflexes equal and normal.

Laboratory Examination: Urinalysis negative. Blood negative.

X-ray reveals no cervical rib.

Diagnosis: Scalenus anticus syndrome.

April 27, 1934: Condition about the same. Scalenus anticus resection advised but operation refused.

CASE IV. M. R., white female, aged seventeen years, single, house work.

April 21, 1934: Admitted to Tulane Hutchinson Memorial Clinic.

Chief Complaint: Pain and weakness of right forearm.

Present Illness: Seven years ago struck by moving automobile and fell on right arm. Her physician told her she had "greenstick fracture" of forearm, undetermined whether radius or ulna. Since then has continued to have dull, aching pain over ulnar aspect of forearm just below elbow. Sometimes pain, throbbing in character, frequently radiates down to wrist and up arm to shoulder and neck in supraclavicular region. Occasionally pain is on radial aspect of forearm with radiation downward. Pain is

sometimes absent and on other occasions becomes very severe. Has also had weakness, especially noticed on lifting objects and in writing. When arm is in dependent position, becomes cold, numb, and patient experiences tingling sensation, especially in fingers. Carries arm in sling in order to obtain relief.

May 14, 1934: Examination reveals tenderness over course of radial, ulnar, and median nerves in right forearm. Less strength in grip of right hand than left. Tenderness in right supraclavicular region over the scalenus anticus muscle. O.M.G. reveal definite diminution of arterial oscillations when head is extended and turned to right. On left, oscillations are normal. Laboratory examination of urine, blood, and feces negative.

X-rays of the cervicodorsal spine show no evidence of cervical rib.

Diagnosis: Scalenus anticus syndrome.

May 16, 1934: (Tulane Surgical Service, Touro Infirmary.) Cervicodorsal sympathetic ganglion block with novocaine on right side. Horner's syndrome immediately after. However, there is no relief of pain in arm, eliminating cervicodorsal sympathalgia.

May 17, 1934: Operation: Under gas anesthesia through transverse incision over posterior edge of sternocleidomastoid muscle in the right supraclavicular fossa the scalenus anticus muscle is exposed. Muscle is thickened and apparently spastic. Muscle divided just proximal to its insertion on the first rib. This is followed by sudden release of the first rib and its disappearance into the depths of the wound.

May 24, 1934: Convalescence uneventful. Patient relieved of all symptoms. Discharged from hospital.

May 28, 1934: Free of all previous symptoms. Has some hyperesthesia along distribution of superficial cervical plexus. O.M.G. show increase in oscillations as compared with the preoperative findings (Figs. 5, 6).

June 18, 1934: Patient complains of some pain in right arm and also in pectoral region.

November 17, 1934: Patient still has some pain in the right supraclavicular fossa which radiates down the arm. Tenderness over the old attachment of the scalenus anticus muscle. Pain and tenderness much less marked than that experienced prior to operation. Patient is able to use upper extremity, which previously had to be supported in a sling.

COMMENT: Because of the complete relief of symptoms for a month following operation and recurrence of symptoms after that time we believe that the gap between the ends of the divided muscle has been bridged by scar tissue. This possibly is causing a renewed pressure on the subclavian structures.

Based on this experience we have concluded that simple division of the scalenus anticus muscle is probably not sufficient, but that resection of the distal portion should be done in order to obviate such a recurrence.

CASE V. S. G. D., white female aged twenty-seven years, married, housewife. July 17, 1934: Admitted to Tulane Hutchinson Memorial Clinic.

Chief Complaint: Constant dull aching pain in right supraclavicular region, shoulder and arm.

Present Illness: About three months ago began to notice heavy feeling and prickling sensation in right arm and hand. No limitation of motion. No history of trauma. Patient complains of rather constant dull, aching pain in right supraclavicular region, which extends over the shoulder and into the arm.

Physical Examination: Temperature, pulse, and respirations normal.

Sthenic habitus, fairly well developed and apparently in good health. Definite exquisite finger point tenderness over right supraclavicular region in area of brachial plexus, none on left side. Pressure on right side over scalenus anticus produces marked diminution in radial pulse on right side but not on left. When pressure is applied on right side, patient complains of pain extending down the ulnar surface of forearm to ring and little fingers. This is absent on the left side.

Laboratory examination negative.

X-ray examination: Anteroposterior and lateral roentgenograms of cervicodorsal spine show no evidence of cervical rib. *X-ray* of both humeri negative.

Oscillometric tracings in right arm reveal definite diminution in oscillations when head is turned and on deep inspiration. When pressure is made over right supraclavicular region the oscillations are almost absent. These findings are not present on left side.

Diagnosis: Scalenus anticus syndrome.

August 17, 1934: (Tulane Surgical Service at Touro Infirmary.) Operation: Under ether anesthesia transverse incision made approximately 4 cm. in length and approximately 2 cm. above right clavicle. Scalenus anticus muscle exposed and found to be very large, in fact, larger than the sternocleidomastoid muscle. Approximately 3 cm. of the distal portion of the muscle was resected. Following resection of the muscle the first rib disappeared into the depths of the wound.

Histologic section of excised muscle showed degeneration and replacement of muscle by fibrous tissue. (Fig. 3.)

August 23, 1934: Postoperative course was uneventful. Patient discharged from the hospital today.

September 24, 1934: Patient states that she has no more pain and is relieved of all symptoms.

October 15, 1934: Patient states that she is feeling fine, having no pain or discomfort. O.M.G. made showing greater oscillations on the right in contrast to observations made before operation, in which the reverse was true. Patient discharged today.

CASE VI. I. J., white female, aged twenty-nine years, married, house work. July 21, 1934: Admitted to Tulane Hutchinson Memorial Clinic.

Chief Complaint: Dull aching pain in left shoulder and left supraclavicular region extending down left arm.

Present Illness: For the past two months patient has complained of pain in left shoulder, radiating to ulnar side of arm, forearm, and fourth and fifth fingers of hand.

Physical Examination: Definite tenderness on pressure over scalenus anticus on the left side. Pressure over supraclavicular fossa on the left side obliterates radial pulse. Similar pressure on right side does not affect pulse. Definite tenderness in left supraclavicular fossa on pressure and, when pressure is applied in this area, patient complains of pain extending down the ulnar side of the left arm and forearm to the fingers and states that she frequently has similar pain, particularly in the fourth and fifth fingers of the left hand.

O.M.G. show definite diminution in the amplitude of arterial oscillations on the left side as compared with the right. This becomes more marked when the head is turned and at the height of deep inspiration.

Laboratory examination normal.

X-ray examination of chest negative. *X-ray* examination of cervicothoracic vertebrae and left shoulder negative for cervical rib and for fracture, dislocation, or bone pathology.

Diagnosis: Scalenus anticus syndrome. Operation advised but due to illness in family it has been deferred.

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DISCUSSION

Dr. Albert O. Singleton, Galveston, Texas: I wish to confirm what Dr. Ochsner has said. I think it was a stroke of genius on the part of Dr. Adson to explain the anatomy and symptoms resulting from cervical rib. The older operations for the removal of the rib which were very extensive, were also impossible. The remedy was worse than the disease. The influence is not from the rib itself but from the fibrous band which extends from the end of the rib and is inserted into the scalenus anticus tubercle on the first rib. The pinching of the lower cord of the brachial plexus and the subclavican artery between the scalenus anticus and this fibrous band causes the symptoms. I do not feel that it is necessary to resect a portion of the muscle as Dr. Ochsner advises. My experience has been that it is sufficient to cut the tendinous attachment of the muscle from the first rib. The muscle retracts out of the way and the structures which are embarrassed move toward the midline, relieving the cause of the symp-

toms. The operation is very simple, local anesthesia being sufficient for its accomplishment.

My experience with the syndrome without the rib is limited to one case which responded with complete recovery following the operation indicated. It is well known that all cervical ribs do not produce symptoms and I think it is reasonable to conclude that a patient may have the same symptoms without cervical rib.

DR. RUDOLPH MATAS, New Orleans, La.: Drs. Ochsner and Gage have rendered a valuable service by directing attention to the group of symptoms described as the "Naffziger syndrome" and by confirming the fact that the scalenus anticus is chiefly responsible for their causation. In the past, the same syndrome has been associated in some cases with the presence of cervical ribs, but the experience of the authors has clearly shown that this syndrome may and does occur in the absence of cervical ribs, and solely as the result of the compression of the brachial plexus and subclavian artery by anomalies of attachment and development of the scaleni.

In many cases, cervical ribs are present without symptoms of any kind, and are accidentally discovered in the course of *x*-ray examinations of the neck for other causes. In many others, they cause neural symptoms which are extremely variable, but which, in rarer instances, are identical with those described as the Naffziger syndrome. In others, both vascular and neural symptoms appear; and still in others, purely vascular signs are present, which culminate in aneurysm of the third division of the subclavian artery. The fact brought out in this paper that especially interests me is that the neural and vascular signs usually associated with cervical ribs may exist in the absence of these anomalous ribs, and that all these signs and symptoms are relieved by a simple tenotomy of the scalenus anticus. The causal relation of cervical ribs to aneurysms of the third division of the subclavian is too long established to be insisted upon, except to stress *the importance of the scalenus as the chief factor in predisposing to aneurysms of the third division*. These aneurysms usually develop in the segment of the artery that lies immediately outside of the constricting edge of the anterior scalene. Sometimes, the sharp bend of the artery, as it rides over a cervical rib, contributes to the development of the aneurysm, even when the cervical rib is very short and rudimentary, or is continued as a ligamentous prolongation with normal attachment of the scalenus to the underlying normal rib. The presence of a cervical rib or its tendinous prolongation increases the tension of the scalenus anticus and adds to the compression of the artery and plexus. Several explanations have been given for the dilation of the artery on the *distal* side of the scalenus constriction, and by none more satisfactorily than by Halsted and Reid in their now classical work on the cause of the dilation

of arteries on the distal side of partial arterial constrictions; but my point now is simply to stress *the atrophic effect of prolonged partial constriction or compression of the subclavian artery by the scalenes in favoring the development of this type of aneurysm.* There is no doubt that trauma, whether sudden or gradual and prolonged as in special occupations, is an important factor in addition to the predisposing and anomalous scalenal compression. This was clearly demonstrated in the case of a middle aged bricklayer who had a cervical rib and who developed a subclavian aneurysm as a result of carrying a heavy load of bricks on his right shoulder. In the case of a young woman, aged twenty-seven years, on whom I operated last June, the cervical rib had caused no symptoms until she fell and jerked her right shoulder in a tumble down the stairs of a rolling ship. Varied neural and vascular symptoms began to annoy her after that accident, which grew worse with the appearance of an aneurysm the size of a pullet egg, in the subclavian triangle just outside of the edge of the anterior scalene. At operation, the scalenus tendon was divided and the proximal segment of the artery lying under the scalenus and extending from the aneurysmal sac to thyroid axis was found much reduced in caliber, much thinner, softer and flatter from loss of elasticity, caused by the atrophic effect of the prolonged constriction of the scalenus anticus. The division of this muscle completely relieved the constriction. The cervical rib was not disturbed. The aneurysmal sac immediately collapsed on ligating the artery at the proximal pole, precisely where the constricted vessel expanded into the aneurysmal sac. The wound healed per primam, but it was nearly four weeks before the neural symptoms (especially radiating pains in the arm) completely disappeared.

The practice of tenotomy of the scalenus, introduced by Adson, has replaced the old practice of excision of the cervical rib, and its advantages are now generally appreciated; but the fact that the same procedure, when applied to cases in which there is no cervical rib is equally successful, is not generally appreciated.

Dr. Alton Ochsner (*Closing*): About the resection of the muscle, I do not know that it is necessary. When I was serving my apprenticeship in Schmeiden's Clinic at Frankfort-am-Main, I was interested to see the wide resection of the sternocleidomastoid muscle they were using for spasmodic torticollis. Our only reason for suggesting resection was that in one case we had a recurrence. Whether that was due to the formation of scar we do not know, but presume it was. It is well known that cervical ribs in many instances produce no symptoms. They are more frequent on the left side, but are sometimes found on the right. In many cases the rib is on the left side, whereas the symptoms are entirely on the right, the dropping of the shoulder on the right side probably being responsible for the symptoms.

ANEURYSM OF THE TEMPORAL ARTERY

AN ANALYSIS OF THE CASES REPORTED IN THE LITERATURE TO JUNE 1, 1934 AND REPORT OF A CASE OF TRAUMATIC ORIGIN*

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ONE would imagine that the temporal artery from its extensive course along the skull with its proneness to injury would be a frequent seat of aneurysm. Statistical studies on aneurysm, however, have proved this not to be the case. For instance, in a digest of 551 aneurysms published in Great Britain from 1765 to 1847, Crisp mentions only a single example of the temporal (Benson's). Among the 188 aneurysms treated by digital compression that were assembled by Fischer, in 1869, there are only two of the temporal artery (Bouvier's and Mirault's), but this is a mistake for these are the same and not different cases. Of 982 aneurysms following injuries received in war, Simon, in 1917, lists 8, or 0.8 per cent, as arising from the temporal artery. By far the most comprehensive treatise on temporal aneurysm is that by Santi. Therein are collected 40 cases. Inasmuch as the material which has appeared since then has not been correlated, and especially as modern textbooks pay scant attention to the subject, the completion of the history of temporal aneurysm, together with an account of a personal observation, may not be amiss at this time.

REPORT OF CASE

The patient, a negro laborer, was admitted to the hospital June 26, 1925, with two roundish, painful swellings that had appeared several months after the patient had been struck in the left temple with a piece of cement in November, 1923. The skin was broken and the wound bled

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freely, but the bleeding was easily controlled by pressure and the cut healed without untoward incident.

Examination, on admission, revealed a hemispherical swelling just above the zygoma in front of the left ear. It was regular in outline, the size of a hen's egg, transmitted to touch an expansile impulse which was synchronous with the beat of the heart, and on auscultation harbored a bruit.

Anterior to the main tumor and on a slightly higher level was another mass, the size of a pea. This lump presented the same physical signs.

A blood Wassermann test was negative, and a roentgenogram showed no evidence of aortic enlargement.

The diagnosis offered no difficulty. Both swellings were plainly aneurysmal and of traumatic origin. The larger one was located on the superficial temporal artery, and the smaller on the frontal branch of the same vessel.

Therefore, on July 3, 1925, with the patient under ether anesthesia, an incision 1½ inches long was made parallel to and immediately below the zygoma. The parotid gland was freed of its connections until the superficial temporal artery was encountered. The artery was doubly ligated and was divided between the ligatures. Through two small scalp incisions, the parietal and frontal branches were also tied, the latter distally to the smaller aneurysm, and the aneurysms were removed en masse. The man was discharged from the hospital on July 18, 1925, as cured.

COMMENT

This study is based on 108 cases of temporal aneurysm, 107 examples of which were culled from the literature and the observation herewith presented. Nine were spontaneous in origin; 79 traumatic; in 5 the cause was not given, and 15 were of the arteriovenous type. Of the 79 traumatic cases, 41 were consecutive to cuts, 33 to contusions, and in 5 the nature of the injury was not specified. All of the 15 arteriovenous fistulae followed trauma. Temporal aneurysm occurs more often in men than in women. Seventy-five of the patients in this series were males and 13 were females, or roughly a ratio of 6 to 1. In the remaining 20 cases no reference was made to the sex. The right side was affected 32 times; the left, 38 times, and on 39 occasions no mention was made of the side involved. Of the 108 patients, 90 were cured; 2 improved; 1 showed no improvement; 1 died, and in 14, the result was not recorded. Temporal aneurysm is preeminently a disease of the adolescent

and of the young adult, for of the 59 patients whose ages were given, 48 were under forty years of age. The youngest patient was seven years old and the eldest seventy-eight. The greatest number of cases occurred in the third decade of life, namely 25. Operation was performed on 77 cases, with 67 cures; 2 improvements; 1 recurrence, and no deaths. In the remaining 6 cases, the result was not stated. Conservative treatment was practiced 31 times, with 23 cures, 1 death. No mention was made of the outcome in 7 of these cases. The deceased was a patient under Bushe's care. An arteriovenous aneurysm developed after a temporal blood-letting for an apoplectic stroke. The man died nine months after the arteriotomy for the cerebral condition and the fatality had no connection with the aneurysmal disease. In 10 other cases, compression failed and operation had to be done before a cure was obtained. In another case (Stone's), compression had proved of no avail, but before a contemplated operation could be performed, pneumonia supervened, during the course of which an inflammation of the scalp occurred with a spontaneous cure of the aneurysm. In a few cases, operation did not succeed in effecting a final cure until supplemented by bloodless methods or by secondary surgical procedures.

The diagnosis should offer no difficulty, but there is a possibility of temporal aneurysm being mistaken for a cyst raised by an artery (Frestel). In isolated instances pulsation may be absent or so weak as to escape detection. Under such circumstances, the lesion may be taken for an abscess and lanced. According to Velpeau, pulsating hematomas may be confused with it, and Robinson speaks of its close resemblance to a suppurative hematoma. Another disease with which it may be confounded is sinus pericranii. However, the history of an injury, the site of the lesion and its peculiar characteristics should lead to its prompt identification.

Aneurysm of the temporal artery usually originates in wounds produced by cuts, but a blunt force, such as a blow, can give rise to it, or it may occur on rare occasions without any known cause. In the traumatic variety, both the artery

and its satellite vein may be involved in the process. Gunshot wound, as a cause, is unusual. This happened five times, or roughly in 5 per cent of the cases reported. The aneurysm is seated most frequently on the superficial temporal artery.

The prognosis of temporal aneurysm is good. The affection neither threatens life nor endangers health. The lesion, however, is unsightly and annoys the patient by its throbbing. Operation assures a rapid cure.

While non-operative treatment has been used with perfect satisfaction, operative attack has established itself as the means best calculated to effect a safe and rapid cure of the condition. A wide variety of operations have been performed. Some have secured the artery on the cardiac side of the lesion. Others have laid the sac open, have turned out the clot and have tied the artery at its entrance into or preferably at both poles of the sac. Still others have ligated the common carotid artery. This procedure, however, is unwarrantable, because the disturbance to the cerebral circulation caused by ligation of the primitive carotid is fraught with danger. Others have occluded the external carotid artery. While the mortality associated with this operation is low, the percentage of failures is high and it is not to be recommended.

The operation of choice is excision of the sac, because (1) it is easy to do; (2) it is safe, and (3) it offers absolute security against recurrence of the disease. The extirpation is accomplished by isolating and ligating the temporal artery above and below the aneurysm along with any collateral twigs entering the sac, excision of the sac and closure of the wound without provision for drainage.

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DISCUSSION

DR. RUDOLPH MATAS, New Orleans, La.: I fear that most surgeons not specially interested in vascular surgery will look upon a discussion of aneurysms of the temporal artery somewhat as a waste of time. The comparative rarity of these aneurysms, their superficial situation, which makes them amenable to pressure and extirpation or other radical treatment, account for the apparent indifference with which they are regarded in most textbooks. None the less, and especially when they are of the arteriovenous type, they sometimes serve as the starting point of diffuse plexiform or cirsoid tumors which, if not checked in their incipiency, assume a formidable aspect which challenges the best resources of surgery. The mere fact that my good friend, Dr. Nathan Winslow, has thought fit to make these temporals the subject for a paper on this occasion makes me attentive and confident in the expectation of much profit that will derive from his discussion. Dr. Winslow, in his modest and unpretentious way has the habit of digging to the very bottom of any question which he attempts to investigate, and when he is through we know we need go no further for information regarding that particular subject. His analytical and critical study of the extracranial aneurysms of the internal carotid artery is a model of patient, painstaking thoroughness which has made his monograph on the aneurysms of this artery indispensable to all workers in vascular surgery. In anticipation of Dr. Winslow's paper, I have jotted down some unpublished notes of our experiences in New Orleans which may contribute to his statistics.

Aneurysms of the temporal artery are, as a rule, of traumatic origin. They are rare in peace and in war. Until about fifty years ago, when bloodletting by temporal arteriotomy was still in vogue for cerebral congestion, meningitis and apoplexy, aneurysms sometimes resulted from this practice. Their incidence in the collective statistics of Sprunner-Merz show that in a total of 332 aneurysms of all arteries admitted to the Koenigsberg clinic to 1887, 2.5 per cent involved the temporal artery. Simon, in 1917, collected 982 war aneurysms recorded in the general literature of the World War, and of these only 0.8 per cent involved the temporal artery. O. Hahn, in 1921, found that in a series of 195 aneurysms assembled from Kuttner's war experience, 1 per cent involved the temporal artery.

The records of the Charity Hospital of New Orleans from August, 1905, to

July 1, 1934 (twenty-nine years) which I have recently compiled, show 234 operations for aneurysms performed by 37 operators, and of these only 3 involved the temporal artery, an incidence of about 1.2 per cent, including one arteriovenous aneurysm in my service and 2 arterial operated on by other surgeons.

In my collection of 605 personal operations performed on the surgical arteries in forty-six years (1888–1934) *there are 3 arteriovenous aneurysms of the temporal vessels, or an incidence of nearly 0.5 per cent.*

In addition to the 6 cases, there are 2 others, one arteriovenous (L. H. Landry) and one arterial (J. A. Danna), in all 8 records, of which 5 were arteriovenous and 3 arterial, which I will present to Dr. Winslow for his records.

All these cases were caused by direct traumatism (fist and other blows, flying bodies, stabs, buckshot, etc.). They were treated by various methods: extirpation with local ligations, obliterative aneurysmorraphies, and in one an extirpation was preceded by ligation of the external carotid.

There is one class of temporal aneurysm with which we are not familiar here, and that is the aneurysms that result from *rapier slashes* inflicted in students' duels at German universities (*Mensur Verletzungen*). Karl Erb and Irwin Hahn recently reported* three such duelling aneurysms caused by rapier slashes, which occurred at the University of Konigsberg in the years 1930–31 inclusive. In one case, the aneurysm appeared fifty-five days after the injury; in another, in fourteen days; and in a third, six months after the injury. These aneurysms are usually extirpated before they attain large size. They are usually well circumscribed and encysted pulsating tumors which vary from the size of a small bean to a large cherry. According to these authors, duelling aneurysms of the temples were described as far back as 1846 by Burkhardt, and since 1898 by Manz and others.

It is probable that temporal aneurysms are not frequent because the bleeding arteries are usually tied at the time of the injury. When the temporal arteries are *completely* divided, the injury is not likely to be followed by aneurysm. Partially divided temporals bleed persistently and are more difficult to control by pressure than when completely divided. This fact was well known in the days when temporal arteriotomy was in vogue. There is perhaps no bleeding that is more readily controlled by direct pressure than that of a *completely* divided temporal artery. This peculiarity was strikingly illustrated by an episode which occurred in the Charity Hospital about sixty-eight years ago, when Dr. Andrew Smyth was house surgeon. His name is now famous in surgical literature as that of the first surgeon to successfully ligate the innominate artery. According to the story which I heard, in later years, from the interne who had been chiefly

* *Zentralblt. f. Chir.*, 58: 2610, 1931.

concerned in the case, a man was brought in with a bad bleeding cut in the temple. The interne dressed it with a pad and light bandage. The next day he removed the bandage and the wound started to bleed again. He then put on another tighter bandage and thought that would be the end of it. When he removed the bandage, the bleeding started again. This kept on and finally the interne reported to Dr. Smyth, who told him to go back and boldly cut the artery through the wound to the bone and then put on his compress. This struck the interne at the time as very extraordinary advice but he did as he was told and from that moment the bleeding stopped and the wound healed without further interruption.

Dr. William D. Haggard, Nashville, Tenn.: I wish briefly to refer to one case of aneurysm of the temporal artery which I operated upon. It was the result of a gunshot wound with bird shot throughout the face and eyes, with complete blindness. There was considerable pain, swelling, bruit, and expansile pulsation. Under local anesthesia I ligated the temporal artery and its satellite arteries and veins.

In a group of nineteen hospitals at the Mesves Hospital Center, A. E. F., we were able to collect 9 cases of various arteriovenous aneurysm. Previously I have reported one case of enormous cirsoid aneurysm of the scalp which was cured by ligating the left temporal and all the satellite veins on the side and top of the head with entire and complete cure.

Dr. Joseph A. Danna, New Orleans, La.: I wish to mention one case to insert in these statistics. A nurse was telling me some of her troubles one day, and she attributed her not being very strong or very well to the fact that she had had a severe injury to the head a good many years before from the effects of which she had never quite recovered. She said she still had a "throbbing." I casually placed my finger on her skull and found an area about 4 cm. in diameter which was throbbing and seemed quite tense. I told her she must have had a severe injury, that she had a bone defect in the skull, and that she had better let me do something for her. It was a serious thing and I told her it would probably grow worse. About six months later she said, "You were fooling me about this head of mine, why did you not tell me I have an aneurysm?" She told me then that a rival of mine had examined her and said she had an aneurysm. I looked at it again, and by placing my finger on one particular spot all the pulsation stopped. There was a bone bottom to the thing. I said, "You have an aneurysm. We can operate under local anesthesia and you will be all right the next day." She agreed and under local anesthesia I exposed the aneurysm through a much smaller incision than the aneurysm itself, made a small incision through the venous wall, put in three fine silk sutures in the arteriovenous opening, reinforced the suture line by tacking the wall of the vein to it by two more sutures. Then closed the venous opening completely, and closed the scalp wound with one little silkworm suture. She made an uneventful recovery.

RIGHT PARADUODENAL HERNIA

ROBERT C. BRYAN, M.D., F.A.C.S.

RICHMOND, VA.

V. E. will be nineteen years of age on June 13, 1933. She is the oldest of four children and has always been thin and considered delicate by the family. At three years of age she had measles; at five and one-half years diptheria; at six and one-half years influenza; at seven years she had pneumonia and ptomaine poisoning and was very sick about eight weeks. At ten years she had "trench mouth." Occasionally she has had attacks of pain in the "stomach" high up, the pain being worse on the right than on the left side. This pain was always brought on by eating a heavy meal (usually in the evening) and for which she would lie down, take aspirin and occasionally a cathartic. She had noticed on many occasions that her "stomach" would swell after eating, so that she would loosen her clothing. She had never worn girdles or any constriction about the waist. She had never noticed that her abdomen would become more swollen on one side than on the other, although her grandmother stated that she had noticed the right side more prominent, and the left sunken in. The grandmother likewise volunteered the information that the patient "threw up" a great deal as a baby and would cry after eating, and they had found out that the most comforting position was to put her on her stomach. The family has always regarded her as having "chronic stomach trouble." She was accustomed to taking a gentle laxative two to three times a week. She has never taken any constant exercise as it always brought on pain, and violent exercise, more pain. Swimming was her favorite exercise but she was afraid to dive for fear of hitting on her stomach. She attributed the present attack to roller skating, although in her mind she has always associated the attacks of pain as having something to do with her periods. The attacks have been very frequent during the past fall and winter.

Examination. February 10, 1932. The patient is tall and thin. The teeth are in good condition. There is some tonsillar tissue. The heart is negative, blood pressure 110/80, lungs negative, temperature 98°F., pulse 100, respiration 20. The abdomen is slightly asymmetrical, being more prominent on the right, and there is moderate rigidity of the right rectus. Pressure over McBurney's point produces pain, not only here but higher up, apparently about the gall-bladder region. There is likewise pain on sigmoidal compression, and tenderness at the midlumbar point. She is menstruating,

but does not on this occasion associate her periods with the colicky pain in her abdomen. There is no nausea, and her bowels had moved in the morning, although unsatisfactorily. The diagnosis of appendicitis was hardly in strict keeping with the physical findings; however, a moderately acute abdomen was recognized and operation was recommended.

She was taken to Stuart Circle Hospital and prepared for operation. The following laboratory reports were made: Urine (uncatheterized): alkaline reaction, albumin and sugar negative, occasional pus cell and red blood cell, few epithelial cells, mucus. Blood: W.B.C. 7575, neutrophiles 83 per cent, lymphocytes 11 per cent, leucocytes 3 per cent, basophiles 2 per cent, Turck's irritation 1 per cent. Hemoglobin, 92 per cent. Coagulation, two minutes. Blood sugar, 118 mg. Kahn reaction negative.

Operation. Under ethylene anesthesia, a right gridiron incision was made. The cecum bulged forward as if there were behind it a very large retrocolic cyst. Due to this retrocolic pressure the mesentery was shortened so that the caput was presented in the wound with some little difficulty. The appendix was located; there was no gross disease and it was removed. The investigating finger in the pelvis found the uterus normal and in good position; the right ovary slightly larger than normal, and the tubes normal. There was some clear peritoneal fluid running back up in the wound. During the investigation it was noted that the usual festoons of the small intestine were absent, and gentle delicate cobweb adhesions were felt in the midline, which were easily separated. They were apparently between a few loops of the small intestine and the parietal peritoneum.

The real pathology was evidently higher in the abdomen. The muscles were now cut at right angles and the incision extended upwards. The wound being opened up better with wide retraction, the bulging, elastic, symmetrical, retroperitoneal tumor could be readily identified as an abnormal state and became more prominent as the superimposed pressure had been removed. This tumor extended as far as the caput inferiorly, had compressed and dislodged the ascending colon outwards, reached as high as the lower pole of the kidney, and extended internally to the borders of the vertebrae. It was ovoidal, probably 10 inches long and 5 inches wide, and its anterior margin was apparently touching the parietal peritoneum. Some large mesenteric lymph nodes, the size of small grapes, were palpated between the mesenteric leaves. The searching finger felt the sharp margin of a well-defined foramen up under the transverse colon and omentum in the midline. On better retraction and the transverse colon thrown upwards, this opening was seen to be about the duodenojejunal fossa, and into which there had been an invagination of the intestine. Two limbs of the jejunum were disappearing into the hole, which even then would easily admit the tips of two fingers. The intestine was smoothly

and rapidly pulled back into the abdominal cavity. It was whitish, anemic, no adhesions whatever, and was otherwise normal. I fancy some 10 or 12 feet of the jejunum were so put back into the abdominal cavity proper. The mesentery appeared unduly long, with here and there dilated veins and patches of a purplish color. The finger, put in the sac to estimate more accurately its dimensions, showed the cavity well defined; no adhesions or bands; the walls slick and reaching upwards as high as the kidney; outwards, beyond the margin of the ascending colon's outer wall, the mesentery of the ascending colon being shoved aside by the encroachment of the tumor from within. The lower margin of the sac could not be reached, but had gone as far as the ileocecal junction, which had produced the undiagnosed bulging of the caput and its abnormal forward displacement which has been referred to. The finger, now pointed forward, was apparently covered with but a single layer of the thin glistening peritoneum. The margin of the opening was well defined, firm, sharp, fibrous and as large as an egg cup. At this point the patient became somewhat shocked, so the head of the table was lowered and stimulants given, and we had but little time left for closure of this very well-outlined opening. A rather large artery served to mark the right boundary of the hole. Its pulsation could be readily felt, and it was lying 2 or 3 mm. from the free edge of the foramen. Three equidistant sutures of 0 catgut were used in the attempted closure of the rim of this aperture. The remaining small holes, so left between the sutures of the partially closed fossa, were scratched with the scalpel, with the hopes of encouraging uniting granulations later on. The opening was not done away with entirely, or as well as we should have liked, as there were now four little foramina rather than one large one. Dr. Beck's report on the pathology was "the appendix is 4.5 cm. × 5 mm., diagnosis, subacute on a chronic catarrhal appendicitis."

The patient had an uneventful recovery and left the hospital on the thirteenth day.

Fossae about the duodenojejunal junction have always excited considerable anatomical and surgical comment, and although the writer has had no opportunity other than in the case herewith on which to base an opinion, it appears that the embryological development and anatomical sequence substantiate the statement that all fossae, dimplings, and hernias in this region are congenital and not acquired.

In considering the many dimplings of the peritoneal cavity about the duodenojejunal angle which have been dignified by different names, it is evident that an intimate study of the

embryological development of the peritoneum, rotation of the intestines, their umbilical escape and later recession, the development of early embryological structures, the enormous mesonephros and its ultimate disappearance, the thorough disproportion in early uterine life of the greatly enlarged liver, the growth, elasticity, resilience, fusion of its several layers, and delicacy of the peritoneum, are some of the features which must be considered from a direct anatomical and embryological standpoint to appreciate the ultimate development of these fossae. When one recalls that in early uterine life the disproportionate size of the mesonephros and liver causes them to occupy the abdominal cavity to such an extent that the intestinal tract for its growth must seek a more comfortable bed elsewhere, and is actually squeezed into the umbilical stalk, we realize the lack of space for this growing, developing tube which returns along the end of the second month into the abdominal cavity. In a series of investigations of pig embryos (about the third month) the anemic, small intestinal cord, no larger than a thread, is found in the right flank compressed and flattened by the adjacent abdominal organs.

It seems interesting that in the many books which dwell upon embryological development and intestinal rotation so little attention has been given to the peritoneum.

The intestinal tract is at first a straight tube and later conforms to zoological laws of rotation and uniform localized ballooning to form the stomach and colon and accommodate itself to the abdominal cavity. The gradually lengthening mesentery of the peritoneum, anchoring the intestines to the back bone, early assumes the duty of permitting of the inviolable laws of nature in the rotation of the intestinal tract to the right. It is interesting here to question why the caput falls to the right so uniformly when this part of the abdominal cavity is so much taken up by the greatly enlarged embryonic liver. Broesike, in explaining this phenomenon, says it is produced by the adhesion between the hepatic flexure and the left side of the duodenum, which in the blending of the latter

to the right, gives the original impulse to the movement of the cecum and ascending colon to the right side of the abdomen. It may be that in a hesitant decision, Nature permits of a

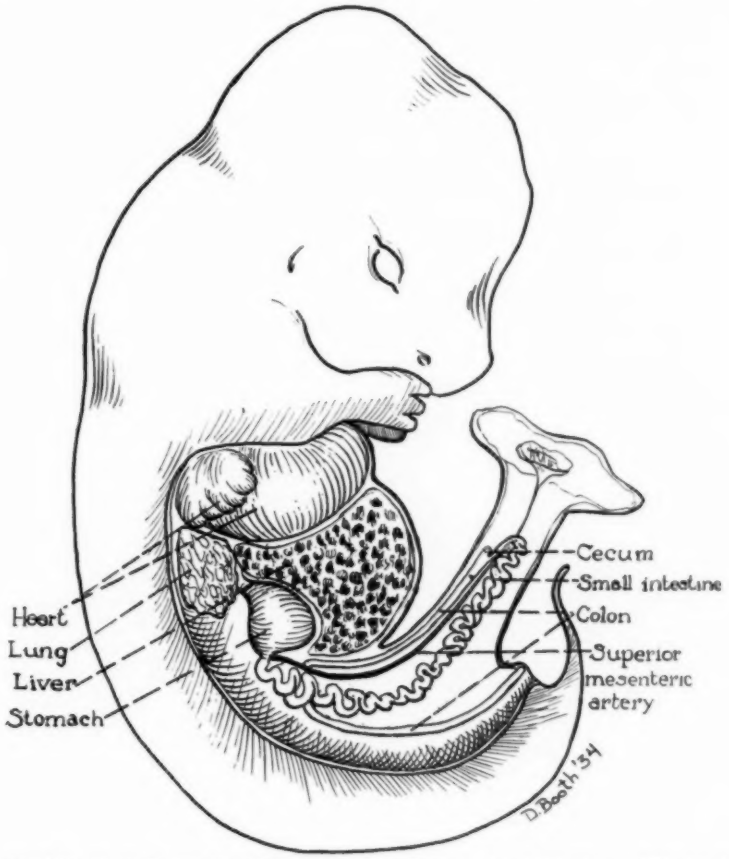


FIG. 1. Umbilical stalk, and lack of abdominal space for intestines. (Pig embryo.)

heterotaxia or transposition which allows of a more comfortable adjustment of the intestinal tract in the left flank. No satisfactory explanation has ever been given why in the twelfth week, in the falling of the mesenteric leaf to the right, there is a fusion of this layer of peritoneum with that lining the posterior abdominal wall, so that there is ultimately a disappearance of one of the leaves. A more positive and deciding factor to be considered is, that when these two peritoneal layers fail to amalgamate, a congenital diverticulum forms to the right with

the mouth of the sac appearing above at the duodenojejunal junction.

We do not propose to consider in any way the manifold

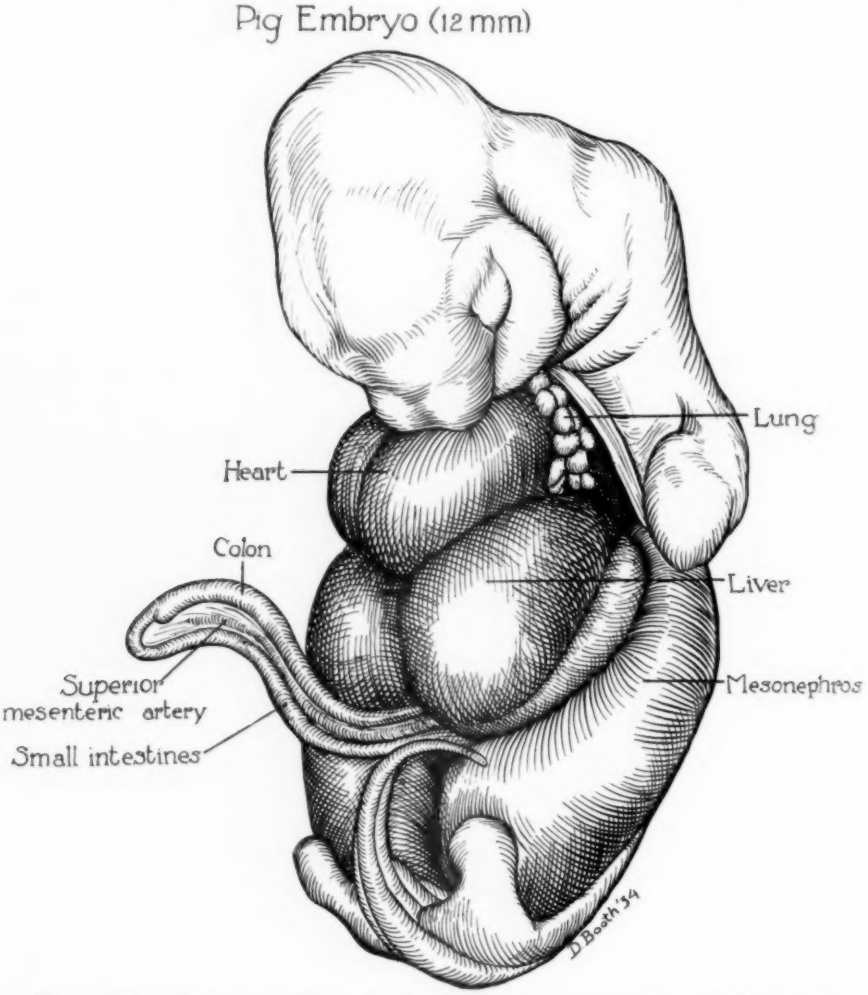


FIG. 2. Mesonephros and liver, further forcing intestines into umbilical stalk.

fossae which occur elsewhere in the peritoneal cavity, such as about the caput coli, the sigmoid, inguinal, femoral, umbilical, through the foramen Winslow or about the female generative organs, but to limit our discussion to the fossae about the duodenojejunal junction, particularly those to the right.

The first point that engages our interest is the anatomical

fact that the duodenojejunal junction of the intestinal tube selects an acute angle to shove its way through to the left under the superior mesenteric artery, which structure, if placed higher

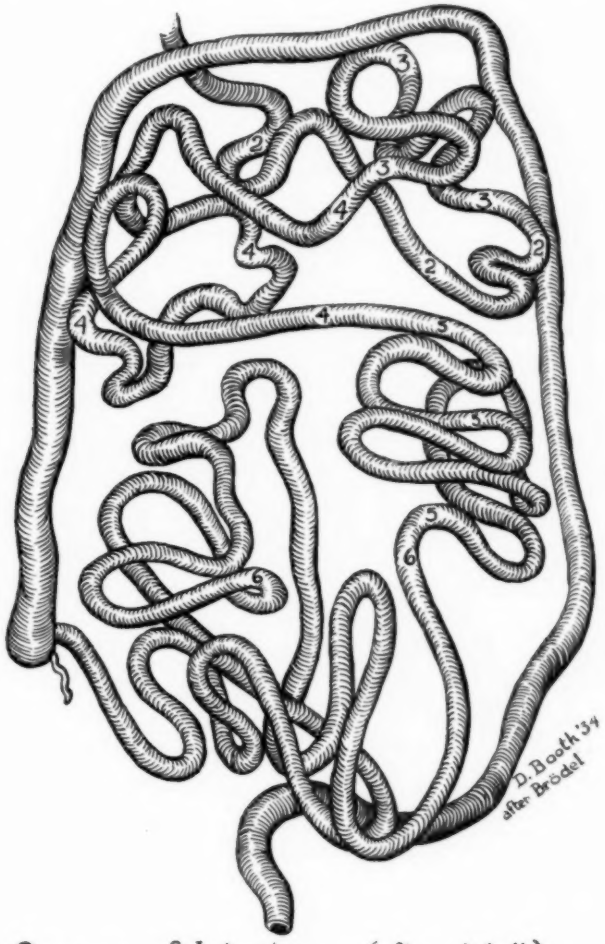


FIG. 3. Schematic adjustment of intestines in abdominal cavity, alternating right and left. (After Mall.)

or lower than the usual, more to the right or left, of larger caliber, or more acute angle, must necessarily offer mechanical obstacles and fascial embarrassment to the successful emergence of the intestine to the left of the median plane. The duodenum, too, may be more redundant or fixed, according

to the development, attachment, or actual elasticity in the fibers of the ligamentum suspensorium duodeni. This, we take it, is the key note, the anatomical explanation of that very rare and exceptional class of cases in which the duodenum does not enjoy a satisfactory and normal emergence to the left, and thus leaves the intestine, the jejunum in particular, imprisoned in the right iliac fossa. In other words, we presume to state that the more reasonable explanation of right retroperitoneal hernia is invariably, and in every instance, congenital and not acquired. It has been stated that some of the causes of such paraduodenal herniae are: the constant fluid contents of the intestines; the rocking, shaking and jiggling of babies thus tending constantly to further dimpling of the duodenojejunal fossa, and bringing about an acquired hernia such as one sees in later life in ventral, inguinal and femoral herniae. Rather a peculiar and interesting fact is that the transverse colon and omentum have never been found in paraduodenal hernia, and since the mesenteric attachment of the small intestine is directed slightly to the right, the wave of impact from shaking, caressing and jiggling the babies would tend by vertical posture, gravity, and the normal transmission of this force, to develop inguinal or femoral hernia by directing the intestine away from the duodenojejunal fossa rather than increasing the size of these dimples by this means of quieting the infant, unless we imagine the baby being so quieted upside down.

Halliwell, in discussing his case, says in support of the congenital theory of paraduodenal hernia:

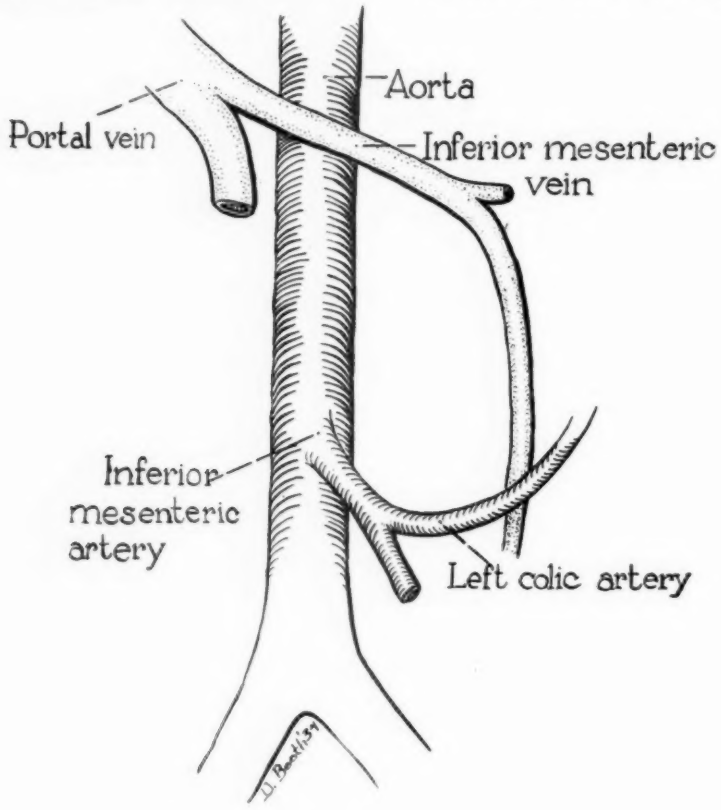
> It is difficult to imagine a force which will continue to act until the small intestine has been segregated into a sac, leaving the abdominal cavity empty. There are many similar folds and fossae in the abdominal cavity, some of which are larger than the duodenal fossa and are practically never the site of a hernia. Apparently, what limits the escape of the entire small intestine into the pocket is the mechanical explanation of too great traction on the mesenteric attachment.

If one were to throw a handkerchief over a piece of stretched string (peritoneum over the intestines) and then allow the

string to collapse and rotate off to one or the other side at any given angle, the folds, fossae and reduplication of the handkerchief just at this angle are seen never to be the same. This may be said of the intestinal tube, its covering by the peritoneum and its rotation about the duodenojejunal angle. Now we have the tense peritoneum more marked above, as it is elevated by the strutting superior mesenteric artery, and now below, protruded by the inferior mesenteric vein, and now to the right, and now to the left, as fascial bands, more highly developed peritoneum, vessels or other impedimenta may happen to exercise their anatomical influence on the gentle physiological rotation just at the duodenojejunal angle. The duodenum, too, having been retroperitoneal, suddenly emerges to become completely invested by the peritoneum, a further reason for reinforcement of the peritoneum at unpredicted sites. Apparently, the production of these paraduodenal pockets is directly dependent upon anatomical conditions. They cannot be foretold, are fortuitous, vary in their size, boundaries and location, and really have but one feature which is constant and uniform; that they are determined and located by the vascular structure found just here, the superior mesenteric artery and the the inferior mesenteric vein, over which the peritoneum is thrown, thus producing pockets and recesses in direct proportion to their course, elevation and prominence. The vascular structures at this location and their relationship is known as the arch of Treitz.

Treitz states that on the left side of the duodenojejunal flexure a peritoneal fold is seen. This fold varies in shape and size. Most frequently it is semilunar, the thin concave edge looking upwards and to the right, and surrounding the bowel at the level of the flexure. The upper horn of this semilunar fold is blended with the inferior layer of the transverse mesocolon, and especially at the point where the inferior mesenteric vein passes beneath the pancreas. The larger lower horn is continuous on the inner side with the peritoneal investment of the duodenum, and at the outer end with the peritoneum of the

transverse and descending mesocolon. In the upper horn, at a variable distance from the edge, lies the inferior mesenteric vein forming an arch, with the convexity looking upwards



Arch of Treitz

FIG. 4. Arch of Treitz about which paraduodenal hernias occur.

and to the left. The lower horn is less distinct, composed exclusively of two layers of peritoneum, and, at some distance from its free border, one sees the inferior mesenteric artery and its branch, the left colic artery. From the relative positions of these two vessels there results a vascular arch (always referred to now as the arch of Treitz) which surrounds the fold in question.

Landzert states:

With the newborn, whose vertebral column is without curvature, in the dorsal position, the floor of the open abdominal cavity presents a deep

and wide depression which is bounded superiorly by the pancreas and inferiorly by both kidneys. Towards the left, this depression is divided by a marked and slightly convex longitudinal fold, on the right into a wider, deeper, and on the left, into a narrower, shallower depression. This fold, which may attain a height of one centimeter, contains the inferior mesenteric vein and the left colic artery, and is merely a protrusion (duplicature) of the fold of the parietal peritoneum caused by the position of these vessels. Herein is enclosed a transverse fold for the inferior mesenteric vein, in the form of a continuation of the longitudinal fold towards the right, surrounding the initial portion of the jejunum. There is also the duodenojejunal plica with Treitz's fossa, which exists quite independently of these two folds.

This vascular arch is the point of entry for the great majority of retroperitoneal hernias, since the small intestine is displaced below it from the right towards the left, and distends the above described folds.

Eppinger in the study of 25 cases found the following: In 7 cases the vessels ran in the edge of the fold; in 15 cases the vessels were distant 4–17 mm. from the edge; in 3 cases the vein lay behind the posterior wall of the fossa.

In investigating the subject in the newborn, Landzert showed that a large fossa was formed (separate from, as a rule, but capable of union with, the fossa of Treitz) on the posterior wall of the abdomen, by the raising up of a large fold by the inferior mesenteric vein and the left colic artery. The fold, in fact was practically a mesentery of the vessels. "The fossa," Landzert says, "is bounded above by a fold containing the inferior mesenteric vein, to the left by a fold containing the left colic artery, below by the mesenterico-mesocolic fold, and to the right by the mesentery of the small intestine." The relations of this fossa with that of Treitz are well shown in a transverse section of the abdomen.

On studying the subject, it will be noticed that the number of the fossae described have been increasing. From the one of Huschke and Treitz, the two of Gruber and Landzert, we come to three of Jonnesco. In 1896, two other fossae, a retroduodenal and paraduodenal, were mentioned by Jonnesco in Poirier's Anatomy.

Gruber, in 1859 and 1861, wrote an article on the fossae described by Treitz, and mentioned, as variants, a fossa showing three funnel-shaped divisions and a division of the orifice

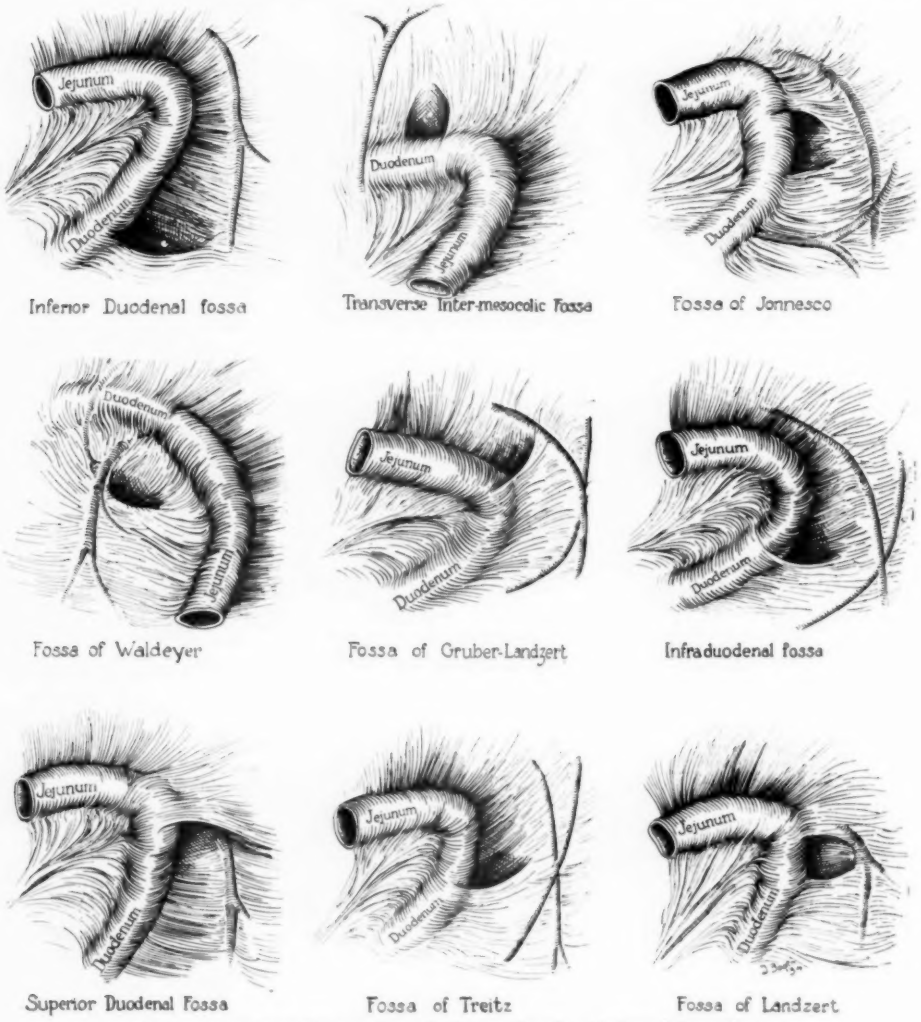


FIG. 5. Various fossae found about duodenojejunal junction.

of the fossa by fibrous bands. Waldeyer, in 1868, found the fossa of Treitz present in 73 per cent of 250 cases consecutively examined, and stated its size varied from the tip of the finger to one holding as much as 12–18 inches of small intestine.

In 1862, Gruber again published a record of cases in which

he described the existence of a right duodenal hernia which he explained as being due to the right-sided position of the fossa duodenojejunalis of Treitz. The duodenum instead of terminating on the left side of the spine was in shape something like the letter s, and its junction with the jejunum lay to the right of the lumbar vertebrae.

It seems reasonable then that a more satisfactory explanation of right duodenal hernia is the embryological failure of the intestine to find its way to the left under the superior mesenteric artery, and consequently it remains in its original position in the right iliac fossa. We must not ignore the evident influence of the vitelline duct in restraining the normal rotation of the intestine at this early embryological period.

As we found in our case, the upper jejunal end failed to rotate at the flexure, and turned very quickly into the hernial sac, and the intestinal mass returned an efferent limb to the abdominal cavity, that is the most proximal part of the umbilical loop. The space for that proximal part of the intestine is made very small through the rapid growth of the liver and mesonephros, yet rotation of the s proximal part of the intestine must take place in that confined space. Should the rotation begin while the umbilical loop consisted of simple uncoiled limbs, rotation would seem simple of execution. If, however, rotation be delayed until the small intestinal limb of the umbilical loop has grown into coils, the proximal coils would find it difficult to rotate in the small wedge-shaped space between the mesonephros. The outer end is free, the inner is confined by the liver anteriorly and each mesonephros posterolaterally. The rotation of the intestinal loop about the superior mesenteric artery as its axis, the subsequent disappearance by absorption, or adhesions to the posterior abdominal wall of that portion of the mesentery suspending the ascending and descending colon, bring about variable attachments and anomalous states.

Moynihan describes the following nine distinct types of duodenal fossae:

1. The superior duodenal fossa lies to the left of the ascending portion of the duodenum. Its size is variable, and adhesions or fat deposits may render it unrecognizable.

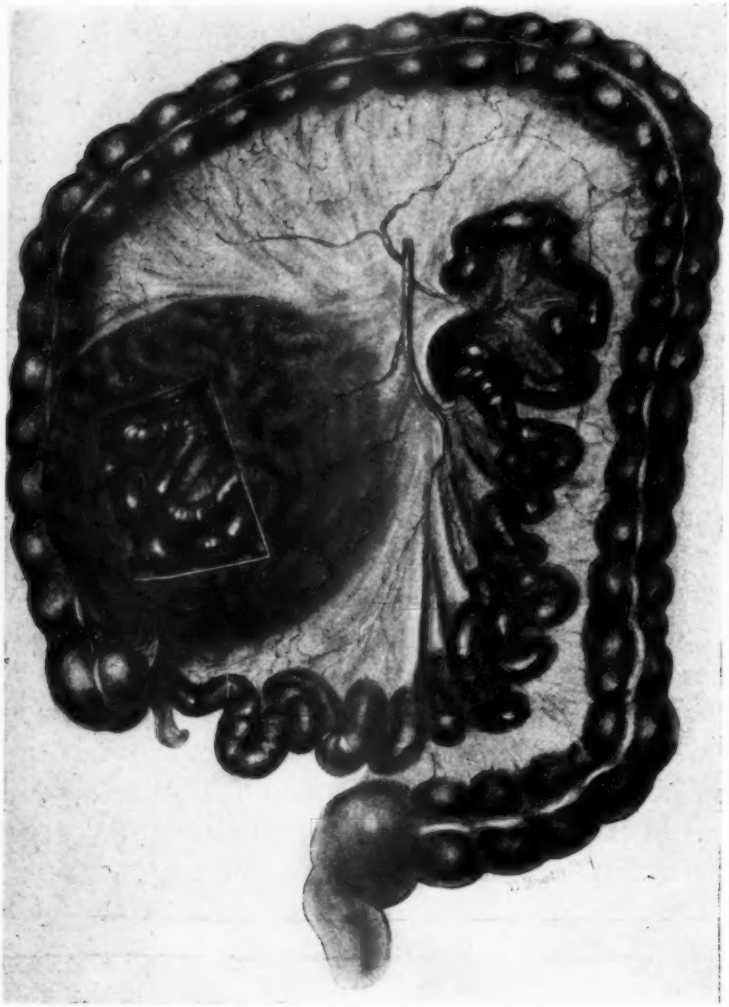


FIG. 6. Condition which occurred in our case, showing superior mesenteric artery along free border of hiatus.

2. The inferior duodenal fossa (fossa of Treitz) is easily located and lies to the left of the ascending portion of the duodenum between the third and fourth lumbar vertebrae.

3. The posterior duodenal fossa lies behind the upper part of the ascending limb of the duodenum.

4. The duodenojejunal fossa lies at the base of the transverse mesocolon.

5. The intermesocolic fossa occupies a position somewhat

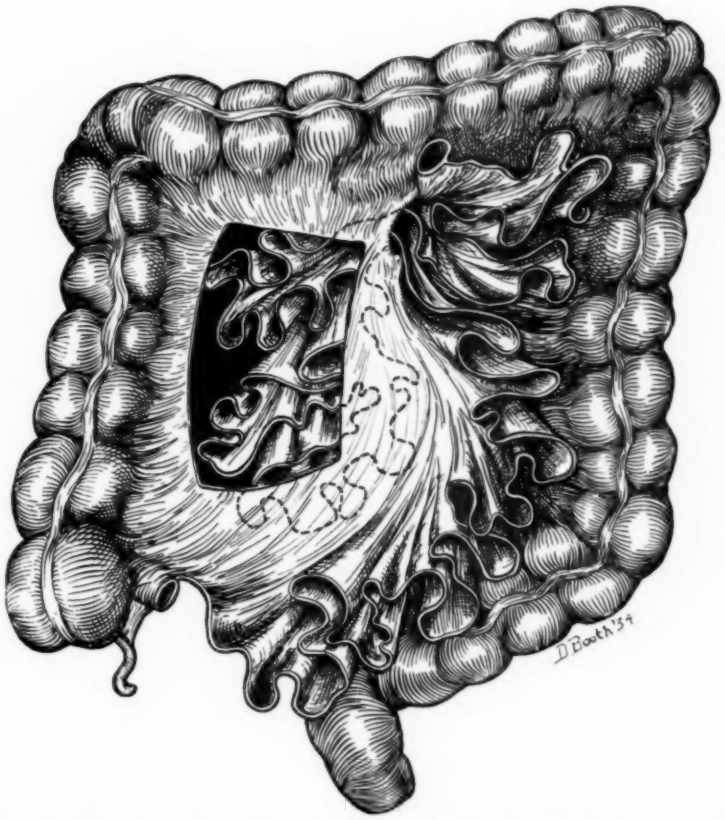


FIG. 7. Direction of mesentery to right, intestine having been removed.

similar to that of the duodenojejunal fossa, in that it lies at the root of the transverse mesocolon, but, unlike the latter, it runs horizontally along the mesocolon.

6. The paraduodenal fossa, of which there are two types, lies to the left of the ascending limb of the duodenum. The inferior mesenteric vein or the superior mesenteric artery may serve as guides in locating these fossae.

7. The intraduodenal fossa may occur in the region of the duodenojejunal angle.

8. The mesentericoparietal fossa of Waldeyer lies behind the superior mesenteric artery and may be found by examining the first part of the mesojejunum.

FIG. 8. Intestine replaced in abdominal cavity, and opening closed by three sutures.

9. The parajejunal fossa also lies behind the superior mesenteric artery and is easily located in the first part of the mesojejunum. It is closely associated with the mesentericoparietal fossa.

Lower and Higgins state that many and questionable factors enter into the anatomy and etiology of fossa formation, and enumerate the following:

1. Traction folds (Nixon).
2. Tense margins of the fossa combined with freedom of movement of the intestines plus peristalsis (Treitz).
3. Embryonic origin, as late descent of the cecum.
4. Failure of the root of the mesentery to unite with the posterior abdominal wall.
5. Formation of pockets during intestinal rotation.
6. Formation of fusion folds in fetal life.
8. Elevation of peritoneum by blood vessels (Waldeyer).
7. Physiologic adhesions (Toldt).
9. Mesoduodenum (remains of early folds) (Treves).

To these may be added the position of the fetus in utero, its turning and twisting thus producing by pressure a dilatation or dimpling of the peritoneum.

It has been likewise brought out that the disappearance of the duodenal mesentery in early embryonic life may result in fossa formation.

Huntington says: "Differences in the rate of growth between the length of the body wall and the length of the mesocolon may play an important part in the production of peritoneal fossae, or small pouches, which in some regions of the abdomen may assume considerable proportions."

He further states that such hernias occur before the obliteration of the primitive parietal peritoneum.

Dr. H. L. Osterud, Professor of Anatomy, Medical College of Virginia, Richmond, has enumerated the following embryologic and anatomic features in the rotation of the intestine about the duodenojejunal angle, and he states that it seems plausible to account for the origin of the retroperitoneal hernia under consideration in the following manner:

> When the umbilical loop was in the process of rotation and the jejunoileal limb was undergoing rapid elongation with extensive coiling, one need only visualize that for some reason certain of the coils of the jejunum,

located toward the proximal end, were prevented from passing under the colonic limb as they have to do. Such passing under the colonic limb would, because of the configuration of the space available for the intestine, be much more certain in the distal part of the loop, but the proximal portion is located between the two mesonephroi. These embryonic organs assume surprising size in the very stages of development here under consideration. The actual rotation of the intestine being dependent upon growth processes might become imperfect because of imperfections in the growth processes themselves. At any rate, it seems reasonable to assume that failure of rotation of some of the small intestine completely under the colonic limb could leave these unrotated coils on the right side instead of coming completely free to the left side of the abdomen. Should this occur it is conceivable that there might be decided interference with the fixation of the cecum and the ascending colon and the root of the mesentery of the small intestine. It is also conceivable that such interference might merely be partial. It may be assumed in the present case that this was so; that the unrotated coils of the jejunum came to lie behind the developing mesentery of the ascending colon, in some way preventing the fusion of this mesentery even with themselves. But fixation of the colon did occur to the right and above these intestinal coils. Fixation also of the root of the jejunoileal mesentery was attained except where this root of necessity had to cross those portions of the intestine which connected the unrotated with the completely rotated coils. Here again fusion was prevented. But at this unfused interval an apparent hole would be present, its anterior boundary the unfused root of the mesentery containing the superior mesenteric vessels, its posterior boundary the posterior wall of the abdomen. Through this hole two limbs of the intestine would course carrying the mesentery with them. This hole would communicate with a sac containing these unrotated intestines, a sac closed by the fusions of the mesentery or colon about the mass of intestines. If, when the intestines were pulled out of the hole, all their mesentery had come out free, there should have been no hole visible from in front. There still would have been a hole in the root of the mesentery behind the intestines. It may therefore be assumed that some fusion of the mesentery occurred at the margins of the hole preventing full return of all the mesentery of the herniated intestines.

Dr. William E. Ladd, of Boston, in reply to my inquiry about paraduodenal fossae wrote me the following:

Regarding your question of the fossa of Waldeyer, it seems to me clearly a congenital affair and entirely dependent on an abnormality of the fusion of the parietal and visceral peritoneum. It seems to me that one has to go

back to the temporary physiological omphalocele which takes place around the fifth or sixth week of fetal life and then consider the return of the intestine from the base of the umbilical cord to the abdominal cavity. In this process, of course, the prearterial segment of the midgut returns to the abdominal cavity first, and returns in the right quadrant, and then in the process of rotation crosses over to the left behind the superior mesenteric artery. At this stage, of course, the midgut, that is, all the intestine from the duodenum to the middle of the transverse colon, is attached to the posterior abdominal wall by only a very small stalk at the origin of the superior mesenteric artery. Now rotation proceeds and the visceral peritoneum becomes fused with the parietal peritoneum giving the oblique mesenteric attachment to the small intestine. This fusion may take place in an abnormal position or become arrested at any stage in its development. An irregularity in its fusion will give a right or left paraduodenal pocket or the mesentericoparietal fossa of Waldeyer or one of the various other fossae which have been described. It seems to me that Moynihan's book and other articles having unnecessarily complicated the condition which fundamentally is really comparatively simple from an embryological point of view. I can't say that I consider any of these malformations so simple from a surgical point of view, but I do feel that an adequately large incision, a recollection of the embryological development, and a delivering of a major part of the abdominal contents outside the wound are certainly a help in clarifying what at first sometimes seems a hopeless puzzle.

This we consider a most comprehensive, clear and satisfactory explanation of the whole question.

PHYSICAL SIGNS AND DIAGNOSIS

Moynihan states:

It is an important question, however, to consider whether there are any physical signs by which we are able to predict with a fair degree of accuracy the existence during life of these forms of hernia. That such a diagnosis might be possible was, I believe, first suggested in an admirable article by Lichenstern in Zeimmsen's Cyclopedia in the following words: "Under favorable circumstance, if the hernia is of notable size, I consider it possible to make a probable diagnosis—not a positive one—but, still one that is based upon reasons. The circumscribed globular distention of the mesogastrium, with retraction of the region corresponding to the colon; the firm, elastic, spherical lump which can be distinctly felt when the abdominal wall is thin, giving the impression of a large, somewhat

movable cyst, and extending from the mesogastrium to the left; the peculiarity that this well-defined tumor always yields a sonorous note on percussion, and clear intestinal sounds on ausculation, also the presence of hemorrhoids and the loss of blood from the rectum in consequence of compression of the inferior mesenteric vein permit, when taken in connection with the subjective troubles indicating chronic disease of the abdominal organs, a probable diagnosis to be made."

Moynihan further says:

The tumor which exists in cases of duodenal hernia may be described, therefore, as having the following attributes: (1) It is limited to a definite region of the abdomen. In left duodenal hernia it lies at first to the left and upper; in right duodenal, to the right and lower part of the abdomen, but in each case spreads finally over almost the whole abdominal cavity. Around the tumor there is an area of depression corresponding to the position of the colon. In size, the tumor may vary. It has been described as being of the "size of a child's head." In Barr's case it was equal to a nine months pregnancy. It is slightly movable, but fixed during respiration. (2) The tumor is marked out distinctly by palpation. On percussion it is always resonant. The degree of resonance varies in different cases, and in different parts of the same tumor. But, a striking feature is that the tumor is a palpable definite, resonant mass. In the center of the tumor, or over its whole surface, may be noticed coils of intestine. The tumor may bear a very obvious relation to the clinical condition of the patient, becoming more tense and prominent, and very much more tender when the symptoms undergo exacerbation. As the symptoms decline in severity, the tumor becomes less aggressive. (3) On ausculation distinct gurgling sounds may be heard anywhere in the tumor.

It is an important aid to diagnosis to remember that owing to the position of the inferior mesenteric vein in the margin of the orifice of the sac of a left duodenal hernia, the radicles of this vein may become enlarged, as in the case of the hemorrhoidal veins, or venous trunks on the anterior abdominal wall may be so increased in size as to form striking features of the case. It is especially noted that the hemorrhoidal veins were very much dilated in Strazewski's case. Lower and Higgins call attention to the dilatation of the veins on the anterior abdominal wall, as well as the hemorrhoidal engorgement.

So far as the symptoms of the hernia are concerned, there is little to be said. The symptoms may have been so slight that little or no attention was paid to them, or they may have been so sudden as to swiftly strike the patient down w en apparently in good health. In the most carefully recorded cases, it is not without some importance to note that the history of chronic slight digestive or intestinal troubles could be obtained. In recording future cases, thorough investigation of the earliest history should be especially attended to.

Short in reporting his case states, "My patient had had 40 or 50 attacks of subacute intestinal obstruction which were in the habit of clearing up after two or three days."

In a few of the reported cases of paraduodenal hernia, *x*-ray examinations have been made, but no particular significance has been given to the roentgen findings in the majority of the cases. Lower and Higgins state that *x*-ray is of little aid in the diagnosis as the hazy outlines are most difficult to interpret.

Exner reviews the literature on roentgen findings in paraduodenal hernia. He states that roentgen diagnosis of both left and right paraduodenal hernia is possible in most cases, and that only by thorough and painstaking examination of the entire intestinal tract, and careful analysis and evaluation of the relationships revealed, can we hope to make the diagnosis consistently. He further states, "Naturally such a time-consuming examination will only be undertaken when one has reason to suspect some abnormality, but I believe that, if the possibility of this diagnosis is kept in mind, the diagnosis will be made with increasing frequency."

AGE AND SEX

Right paraduodenal hernia may be observed at any age. The youngest patient on record was that of Paton, aged three months, and the oldest case was that of a woman, sixty-eight years of age, reported by Rose. From the records, this condition was found much more frequently in the male than in the female.

INCIDENCE AND DIAGNOSIS

In 1925, Brown reported 32 authentic cases of right paraduodenal hernia. Of these, 15 were operated on, with 4 recoveries. The remaining 17 cases were found at necropsy or dissection.

Recent search of literature reveals 42 cases. Of these, 25 were operated on, with 13 recoveries and 12 deaths following operation. The author's case makes the forty-third reported case, and the fourteenth recovery following operation.

AUTHENTIC CASES OF RIGHT PARADUODENAL HERNIA REPORTED SINCE BROWN REVIEWED THE LITERATURE

No.	Reporter	Date	Age	Sex	When Diagnosed	Outcome
33	Lower & Higgins	1925	12	F	At operation	Recovery
34	Deaver & Burden	1929	25	M	At operation	Recovery
35	Flechtenmacher	1929	38	M	At operation	Recovery
36	Taylor	1930	32	M	At operation	Recovery
37	Taylor	1930	24	M	*X*-ray	Recovery
38	Masson & McIndoe	1930	41	M	At operation	Recovery
39	Cofer & Phillips	1930	22	M	At operation	Death
40	Dowdle	1932	24	M	At operation	Recovery
41	Exner	1933	56	F	*X*-ray	Recovery
42	Halliwell	1934	35	M	At operation	Recovery
43	Bryan	1934	18	F	At operation	Recovery

Case and Upson, in 1926, reported a case of paraduodenal hernia, but did not state the type of hernia found. They further state that their diagnosis was made preoperatively, and to substantiate their case published a roentgenogram, which apparently is that of right paraduodenal hernia.

Eitel, in 1926, also reported a case which is apparently right paraduodenal hernia, operated on successfully, but from his description it is impossible to say positively what he found.

Venable, in 1930, reported a case of what was probably right duodenal hernia, operated on successfully.

Exner, in his review of the reported cases, is of the opinion that credit for the first preoperative diagnosis made should go

to Taylor, who made the diagnosis in one of his cases by *x*-ray. This was confirmed by operation.

From my review of the literature on this subject, I have not been able to find a reported case in which a diagnosis of right paraduodenal hernia was made clinically, prior to that of Taylor's.

Halliwell, in 1934, made a partial preoperative diagnosis by *x*-ray, and says that after comparing the roentgenogram in his case with that of Taylor's, a more accurate diagnosis should have been made.

TREATMENT

Treatment may be divided into the methods employed in the acute and in the chronic manifestations of this surgical condition. In the acute conditions of retroperitoneal hernia those phenomena brought out by a strangulation are unrecognized, or diagnosed at the time of operation. If the gut is gangrenous, resection is necessary. It may take but a short time to attempt closure of the mouth of the sac, which is evidently most important to prevent a subsequent herniation. The resection, handling the intestines, closing the sac and the prolonged operation produces a severe grade of shock. Figures indicate that operation for right paraduodenal hernia is particularly hazardous, and five times more fatal than left.

Theoretically, the surgeon hopes for a large foramen and no adhesions to deal with, as the intestine is more easily drawn back into the abdominal cavity proper, there is less congestion to the returning venous supply of the intestine, and the margin of the opening is more redundant and presents more tissue for suture. In the event that the intestine cannot be withdrawn, the neck should be enlarged, great care being exercised not to damage or bruise the important vessels in this location. Further operative work, resection, jejunostomy and closure of the sac must be decided upon as surgical indications justify.

Brown in reporting his case said he was able to turn the sac inside out, ligate it at the neck and remove it.

Halliwell, in describing the operation on his case, says:

There appeared to be three large loops or coils of bowel and no small intestine was visible anywhere. These coils overlapped the ascending and descending colon. The membrane covering the loops was moderately thick and it was evident that the condition was a retroperitoneal hernia. The mouth of the sac looked towards the left and over it the superior mesenteric artery could be felt. An attempt was made to withdraw the bowel but this was found to be impossible due to adhesions of the entering and returning loops at the mouth of the sac. Consequently an opening was made into the front of the sac to investigate the condition inside. There was only one layer of peritoneum and the serous coat of the bowel was completely adherent to the wall of the sac. It could be pulled off without difficulty but left an intestine devoid of its serous coat, consequently the hole made in the sac was closed and the neck of the sac was divided on its inferior aspect, thus freeing the entering and returning loops as far as possible, and the abdomen closed. The patient made ultimately a good recovery.

I am indebted to Dr. Wyndham B. Blanton for having been allowed to see this patient in consultation, also to Dr. H. L. Osterud for his collaboration and to Miss Dorothy Booth for the illustrations.

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DISCUSSION

DR. BRADLEY L. COLEY, New York, N. Y.: I had occasion about six years ago to operate on a patient who gave an interesting history of recur-

rent attacks over a period of years. On one occasion he was sick enough to enter a hospital at Bridgeport, but left before radiographic studies could be made. He finally came to Bellevue Hospital and was thought to have intestinal obstruction, the exact nature of which we could not determine. He presented a mass in the upper left side which was about the size of two fists, and was extremely ill. We operated without definite diagnosis other than that of acute surgical abdomen, but found that we were dealing with a left paraduodenal hernia.* This patient had about 8 feet of very much discolored intestine which was still viable.

I wish to call attention to one point of importance in handling these cases, especially where strangulation occurs. In practically all these paraduodenal hernias, both right and left, an important vessel runs in the neck of the sac, and if in releasing the strangulation, which is not easy, one does not bear this in mind there is a possibility of damaging this vessel. By identifying it and by splitting the peritoneum just lateral to it, we were able to stretch the neck sufficiently to reduce the hernia.

This patient was presented before the Surgical Section of the N. Y. Academy of Medicine five years ago but we have been unable to find him since for further study.

DR. RAYMOND P. SULLIVAN, New York, N. Y.: Just two weeks ago, at a staff conference of the Second Surgical Division of St. Vincent's Hospital, New York City, a review of the various types of "hernia" operated upon between 1923 and 1933 was made. There were 1016 case records studied by Dr. M. C. O'Shea. No case of paraduodenal hernia was noted.

In another review, previously made, of cases of acute and chronic intestinal obstruction covering a five-year period (1928–1933) at St. Vincent's, internal hernia, as such, was noted only four times. It seems probable that some cases of internal hernia, of either type, existed, but were not recognized or reported as such.

The following is a case report of a left paraduodenal hernia: F. M., thirty-six years old, married, truckman, weight 192 pounds, admitted to Second Surgical Division, St. Vincent's Hospital, New York City, at 9:00 A.M. January 13, 1934.

Present Illness: Patient stated that he went to bed the night previous in excellent condition. About 4:00 A.M. (or five hours previous to admission) was awakened by a severe cramp-like pain in abdomen, located principally just below and to the left of umbilicus. Pain recurrent in type. Vomiting occurred with pain several times. States that he has had several similar attacks over a long period of time, last previous attack six months ago.

* Strangulated left duodenal hernia. *Arch. Surg.*, 18: 868–881 (March) 1929.

Says that previous attacks just as severe—but of much shorter duration. Also states that each attack has associated "salty taste in mouth."

Previous History: Medical: negative. Surgical: appendectomy 1925 elsewhere; no drainage, convalescence uneventful. Systemic: G.I., appetite good. No dyspeptic symptoms. Constipation obtains. Must take medicine every day to keep bowels open. Respiratory: chronic dry cough. Cardiovascular: "gets short of breath easily." No palpitation. Nervous system: negative. Genitourinary: denies lues. Has occasional burning on micturition.

Family History: Father died of "Bright's disease." Mother and wife living and well. Has three children living in good health.

Physical Examination: Well developed and nourished white adult, thrashing in bed and talking loudly because of abdominal pain. Local abdominal examination revealed some muscular spasm over entire left side. No distinct masses determined. No distention. Liver not palpable. No evidence of hernia in either inguinal or femoral canals. No hernia in wound of appendix scar. No external evidence of injury. Head and neck: negative. Chest (*a*) lungs: few sibilant râles at both bases posteriorly; (*b*) heart: negative. Blood pressure, 142/88; temperature, 99°F., pulse 70, white blood cells, 7300, polymorphonuclear neutrophiles, 82 per cent; later white blood cells, 10,200, polymorphonuclear neutrophiles, 88 per cent. Flat *x*-ray plate showed some fluid level. Impression: probable intestinal obstruction of unknown cause. Operation was deferred at first, but pain and distress persisted with vomiting, ascending pulse rate, temperature and leucocytosis. Exploratory operation advised and performed by Dr. John W. Lawler. When patient was under anesthetic a large globular mass located in abdomen just to the left of the umbilicus was seen and felt. Abdomen opened by straight incision through left rectus muscle. No free fluid present. Some coils of small intestine found collapsed. Globular mass previously mentioned found to consist of coils of jejunum caught in a pocket of peritoneum in left upper quadrant. To determine more clearly the exact condition the entire small intestine was delivered out of abdominal cavity. In doing so some torsion of the mesentery of small intestine was corrected. The parietal peritoneal pocket was emptied of the contained jejunum. This pocket was found to be formed by the inner leaf of the mesentery of the descending colon, which was fused to the root of the mesentery of the upper jejunum, for a distance of 3 inches. It extended upwards from the mid abdomen to the ligament of Trietz. The pocket was everted and its margin sutured to obliterate the fossa. Vessels identified but not disturbed. Intestine replaced in abdominal cavity and wound closed without drainage.

Postoperative Diagnosis: left paraduodenal hernia.

Postoperative Course: Smooth at first until complicated by passage of ureteral calculus on left side which was removed by cystoscope (Dr. T. F. Howley, January 25, 1934).

Discharged from hospital February 15, 1934.

Follow-up: Patient seen and examined November 22, 1934, ten months postoperatively. Wound firmly healed. Bowels moving regularly. Reports no return of symptoms either intestinal or urinary.

Dr. R. C. Bryan (*Closing*): It is interesting to note that both Dr. Sullivan and I attempted to close the aperture in the same way. Dr. Sullivan's case occurred on the left side, of which 162 are reported. Mine was on the right side, of which only 42 are reported.

PSEUDOMYXOMA PERITONEI

REPORT OF CASE PRESENTING BRONCHIAL FISTULA

J. Duffy Hancock, M.D., F.A.C.S. and
Harry M. Weeter, PH.D., M.D.

LOUISVILLE, KY.

INTRODUCTION

PSEUDOMYXOMA peritonei, sometimes called "mucous ascites," may be briefly defined as a disease characterized by the presence of gelatinous pseudomucin in localized masses or diffuse layers within the peritoneal cavity following rupture of a pseudomucinous cyst of the ovary or a mucocele of the appendix, the masses or layers being attached to some intraperitoneal structure or the parietal peritoneum.

It was first described in 1884 by Werth who clearly established the relationship between "myxomatous change" of the peritoneum and pesudomucinous cysts of the ovary. While the condition is not one of the rarities of surgery it is, however, unusual and always of some interest. None of the reports to which we have had access describes an extension of the disease beyond the abdominal cavity. The closest approach are a case reported by Masson and Hamrich in which the diaphragm was "practically destroyed" after invasion of the diaphragmatic peritoneum and one mentioned by Wilson where a probable metastatic area was found in the right lung at autopsy some years after the primary operation for the removal of a proliferating ovarian cyst.

Our case, observed for several years through two operations and an autopsy, showed direct extension through the diaphragm into the lung with the establishment of a bronchial fistula. If this is not unique, it is at least rare enough to justify a review of the disease and report of this particular case.

INCIDENCE

Since the disease may follow the rupture of a mucocele of the appendix as well as a pseudomucinous cyst of the ovary, it may occur in either sex, the incidence of course being much higher in the female. It is most common in the fifth and sixth decades of life and is very unlikely to occur before the age of thirty. Single women are rarely affected.

SYMPTOMS

The symptomatology is rather indefinite. Perhaps the most usual complaint is a rather rapid enlargement of the abdomen. While a sudden sharp pain may occur early, probably as a result of the rupture of the cyst, there is usually only some degree of uneasiness or discomfort resulting from pressure and causing a bearing down feeling in the pelvis, urinary frequency, or shortness of breath. Gain in weight is occasionally observed but some degree of emaciation is the rule. The menstrual habit is usually unaffected. Fever is not to be expected unless there is some degree of acute peritonitis as occurs when a fistula develops between the cystic mass and some portion of the intestinal tract.

PHYSICAL SIGNS

The outstanding physical sign is abdominal enlargement. This distention is usually symmetrical but occasionally several different masses may be identified. The consistency is likely to be softer and more elastic than that noted in an abdomen distended with thin fluid. The impulse palpated when the abdomen is struck with the examiner's finger has been described as a thrill rather than a wave. Vaginal examination shows a rather fixed mass extending in all directions and the fundus does not float as it appears to in ordinary ascites. The skin usually shows some pallor attributable to secondary anemia. Aspiration releases a thick semi-gelatinous fluid which may be colorless or tinged orange to pink.

LABORATORY FINDING

The blood count verifies the presence of a secondary anemia, and the number of white cells will be variable depending somewhat upon the degree of peritoneal irritation caused by the presence of the cystic material. There are no significant urinary changes. The gelatinous material obtained by aspiration or at laparotomy can be identified as pseudomucin rather than mucin by the facts that it is alkaline and does not precipitate on boiling or upon the addition of acetic acid. A reducing substances, glucosamin, has been described.

PATHOLOGY

The mere presence of gelatinous material in the peritoneal cavity does not per se constitute pseudomyxoma peritonei. That disease cannot be said to exist unless masses or layers of this material be fixed or firmly attached to the peritoneal wall or some of the intraperitoneal organs. The original source of the substance is usually the ovary and occasionally the appendix. It is not impossible that other organs might be primary foci. An appendix which is the site of a mucocele has generally undergone previous inflammation and rather complete obliteration of its canal, thus accounting for the fact that only in a small percentage of cases does infection follow rupture. The ovarian lesion giving rise to mucous ascites is a multilocular pesudomucinous cystadenoma. While many large ovarian cysts will present firm gelatinous contents in the loculi at the base of the tumor, the distinguishing characteristic of a pseudomucinous cyst is that its contents remain semi-solid even in the largest loculi, instead of becoming more liquid as the size of the loculi increases. Pseudomucinous cystadenomas of the ovary are usually unilateral (80 per cent) and in only 4 per cent of the cases are they complicated by rupture and the development of pseudomyxoma peritonei. While the gross appearance of the ruptured cysts is usually malignant, Masson

and Hamrich report in a series of 30 cases only 43.3 per cent definitely malignant.

When the gelatinous material is discharged, the pertioneal response may be rather passive or quite active, the latter resulting in general peritoneal thickening, cellular infiltration, formation of connective tissue by actual proliferation of secondary tumors, formation of cysts, or a combination of any of these reactions. The peritoneum may become opaque, velvety, or granulomatous in appearance. The attachment of the implants represents a mild infiltration resembling absorption rather than invasion. When the peritoneal reaction is such that the proliferating cysts are choked at the base they are still likely to rupture and cause other independent growths. The increase in the amount of gelatinous material is probably not due to any myxomatous degeneration of the peritoneum but due to a proliferation of the epithelial elements which are present in the contents of the ruptured cyst and which become attached to the peritoneum and by their proliferation reproduce the original type of cyst. The omentum is a frequent site of involvement and may become quite board-like in consistency. As much as 26 liters of the material have been removed from the peritoneal cavity. The cherry-like color sometimes present is due to capillary oozing.

Microscopically, there are numerous minute papillary growths usually covered by single layered cylindrical epithelium with elongated nuclei situated near the base. Due to pressure, however, the cells may be flattened or entirely atrophied. The cytoplasm is relatively clear.

DIAGNOSIS

A correct preoperative diagnosis is unusual unless some of the aspirated material has been identified. Remembering the possibility of the disease in those cases of abdominal enlargement where the fluid wave is atypical and the pelvis rather fixed will probably lead to more frequent accurate diagnoses.

TREATMENT

The first step in the treatment of the disease is surgical. When the peritoneal cavity is opened, presenting masses of the gelatinous material should be evacuated and the primary focus sought. If ovarian in origin, and the patient is at or past the menopause, a bilateral oophorectomy and appendectomy are indicated; if the patient is much under that age and only one ovary is involved there is some disagreement as to whether or not the unaffected ovary should be removed. If only the appendix is involved, unilateral or bilateral oophorectomy seems rather radical. Hysterectomy, partial or complete excision of the omentum, and even resection of some portion of the intestinal tract may be indicated when these structures show much invasion. Adhesions should be freed and all accessible pseudomyxomatous material removed. This is usually done manually, but saline flushing has been recommended on the belief that saline causes the jelly-like masses to swell and become loosened from their peritoneal attachment. The abdominal wound should be closed without drainage to minimize the possibility of infection. The establishment of a permanent fistula for continuous drainage does not appear to be a good practice. Postoperative treatment by *x*-ray or radium is likely to be of service in delaying or preventing recurrence in some instances.

PROGNOSIS

While frequently neither the implants nor the original focus of the disease are definitely malignant the outlook is not promising. In some instances, the peritoneal growths have been reported as subsiding after the removal of the primary tumor. However, recurrence is the rule especially where the primary site is the ovary. The majority of cases terminate fatally although death may be deferred some years. Recurrences have been reported as early as five months and

as late as twenty-two years. Sepsis and embolism are frequently the routes of exitus.

CASE REPORT

The particular case that led us to present this subject follows:

Miss A. S., a saleslady twenty-nine years of age, was first seen at her residence on March 1, 1930 in consultation with her physician, Dr. F. S. Clark. She complained of abdominal distention, and pain in the lower right side of her chest. Her family history and previous personal history were of no significance with the exception of an attack of pleurisy at the base of the right lung some six months previously. Her menstruation had always been regular and normal, but rather scant.

She had been as well as usual until the previous Christmas holidays when she noticed some vague indigestion which she attributed to overeating. In January, 1930, a rather severe pain was noticed in the region of the liver, resembling the attack of pleurisy several months before. There was a moderate cough at first but this disappeared with the acute pain. Some soreness and tenderness, however, persisted. Two weeks after the onset of pain she observed some distention of the lower abdomen which rapidly increased until the entire abdomen was quite distended. She became short of breath and while not nauseated, could eat only small quantities of food at a time. She had been in bed for the past four weeks.

Physical examination showed marked pallor and emaciation, temperature 101.2°F., pulse 130, respiration 24, and blood pressure 112/88. The lungs were clear but the diaphragm seemed elevated on both sides. Her heart was normal except for its increased rate. Both pupillary and patellar reflexes were normal.

The abdomen was greatly enlarged and tightly distended, the overlying skin glistening. The expected fluid wave could not be definitely obtained. No individual masses were palpable. There was slight tenderness in the region of the gall bladder. The percussion note was flat throughout. Vaginal examination was difficult because of an intact hymen. The cervix was smooth but full, enlarged, and pushed downwards. Pressure upwards on it caused considerable pain. The fundus was small and tightly fixed in a firm mass that filled the entire pelvis. Rectal examination confirmed the vaginal findings and further showed the presence of a small nodule in the cul-de-sac.

Several days later, a drachm of gelatinous material was obtained by paracentesis of the abdomen. The patient was removed to St. Joseph's

Infirmary. Urine examination there was not remarkable. The blood count showed 60 per cent hemoglobin, 3,620,000 R.B.C. and 8600 W.B.C. Dr. Irvin Abell was called in consultation, suggested the diagnosis of colloid carcinoma or pseudomyxoma peritonei, and advised exploration.

The operation scheduled for March 8, 1930 had to be postponed for several days because of a rise in temperature to 105°F. and pulse to 155 with increased tightness in the abdomen. This acute condition rapidly subsided and on March 12, 1930, exploration was done. The left tube and ovary and the appendix were normal. The right ovary was incorporated in a large pseudomucinous cyst which had ruptured. This cyst, including the right tube and ovary was excised. Seven liters of colorless or slightly pinkish gelatinous material were removed from the peritoneal cavity. At no place was it attached firmly to the peritoneum. Even two small masses in the cul-de-sac were easily wiped free. The abdominal wound was closed without drainage. The excised cyst was lost while the gelatinous material was being measured. The latter was soluble in water, not precipitated by acetic acid, but precipitated by 95 per cent alcohol, the precipitate being soluble in water, thus presenting the characteristics of pseudomucin from cystadenoma of the ovary. The patient made a nice recovery and was discharged from the hospital on March 27, 1930.

On April 14, 1930 (two and one-half weeks later) she was readmitted to the Infirmary complaining of pain of one week's duration and a tender mass in the right lower quadrant, nausea, no vomiting and no constipation. Her temperature was 101°F., pulse 104, and respiration 22. She was quite dehydrated and the mass just above Poupart's ligament on the right side was definitely palpable and tender. A diagnosis of localized abscess of undetermined origin was made. Enemas, douches, and local applications of ice gave complete relief and when she was discharged two weeks later, the mass had disappeared, her temperature was normal, and she felt much better generally.

She remained symptom-free for eleven months. In February, 1931, she first observed a feeling of pain, weight, and pulling downward in the left lower quadrant, and then the appearance there of a mass that gradually enlarged to the right and upwards. Her previous menstrual period had been five or six days late and accompanied by slight nausea. She had lost weight and experienced some shortness of breath especially at night. On March 3, 1931 she reentered the hospital.

Her general appearance was somewhat better than at the time of the first admission. The temperature was normal, pulse 90, respiration 18, and blood pressure 125/85. The chest was still essentially negative. Examination of the abdomen showed a large mass entirely filling the left side of the abdomen and extending well towards the right. Tenderness and nodules

were palpable in the left flank. There was flatness to percussion everywhere except in the right flank. The same type of indefinite fluid wave, observed before, could be detected. Vaginal examination showed the cervix normal in size, the fundus could not be palpated and there was a general feeling of fullness throughout the pelvis. A few R.B.C. were found in the urine. The blood count showed 70 per cent hemoglobin, 3,720,000 R.B.C., and 11,300 W.B.C. with 77 per cent polymorphonuclear leucocytes.

A diagnosis of ruptured pseudomucinous cyst of the left ovary was made and the patient's abdomen was again explored. The appendix and left tube were normal. The left ovary showed a large ruptured pseudomucinous cyst. The gelatinous pseudomucin was present in all parts of the peritoneal cavity and in contrast to the findings at the previous operation was firmly attached to the peritoneum in many places, especially in the cul-de-sac and on the omentum, mesoappendix, mesosigmoid, and the peritoneal reflection over the dome of the bladder. The appendix, left tube, left ovary, some of the implants, and all of the easily removable pseudomucinous substances were removed. It was, however, noted on the operative record that "the case appears hopeless as sufficient implants remain to cause further recurrence." The wound was closed without drainage. The pathological diagnosis was chronic appendicitis, chronic salpingitis, and pseudomucinous cyst of the ovary with no microscopic evidence of malignancy, although the clinical and physical findings were definitely to the contrary. Sections through various parts of the tumor masses showed a heavy fibrous stroma; many of the vacuoles contained no epithelial cells, others showed lining cells varying from columnar to flattened cells with no penetration of the basement membranes.

The patient had a stormy convalescence. There was a profuse drainage from the wound, phlebitis in the left thigh, and on one day, there were several incontinent stools of gelatinous material. On April 17, 1931, about six weeks after the operation, she was discharged from the hospital. In June, 1931, a hard tumor mass appeared in the abdominal scar, but never ruptured nor attained much size.

In February, 1932, she came to the office complaining of a soft tumor on the right posterior chest wall at the level of the ninth or tenth rib. Incision into this mass released a large amount of pseudomucinous material.

In May, 1932, she began to expectorate material which she described as similar to that which had drained from the tumor on the back. This I was able to verify later by having her cough some in my presence. The fluid coughed up on three separate occasions was submitted to the laboratory for examination. One specimen gave the characteristics of pseudomucin, the others contained numerous miscellaneous organisms and pus cells and appeared to contain mucus from the respiratory tract.

Chest examination, including fluoroscopy, was indefinite in regard to findings.

In August, 1932, a mass appeared in the right upper quadrant of the abdomen, ulcerated through the overlying skin and discharged principally pseudomucinous material. Later, there was a small amount of fecal drainage.

She gradually became weaker and could not leave the house after October, 1932. In November she developed an edema of the feet and legs. On December 20, she became acutely ill with pneumonia which terminated fatally on December 23, 1932 approximately three years from the beginning of her illness and two years and nine months after the first operations, one year and nine months after the second.

We were able to secure an autopsy and the findings follow.

"The body is that of a tall, poorly developed emaciated white female about thirty years of age. With the exception of the abdomen and back, the body presents nothing abnormal on external examination except the emaciation. In the mid-line scar between the umbilicus and symphysis, there are several firm nodules of varying size. In the right upper quadrant at the anterior axillary line, there is an indurated ulcerating area apparently the external opening of a fecal fistula. On the right side of the back at about the level of the ninth and tenth ribs, there is a soft tumor mass discharging gelatinous material.

"Upon opening the abdomen, it seems that the masses in the old scar are implants, as they do not communicate with the peritoneal cavity. The cavity contains many areas of gelationous material widely dispersed with generalized implants on the bladder wall, fundus, broad ligaments, large and small intestines and under-surface of liver.

"The appendix, both tubes and both ovaries are absent.

"The sinus on the anterior abdominal wall communicates with the colon at the hepatic flexure and the fistula is apparently due to invasion by the gelatinous implant. The sinus on the back communicates with the mass of material at the under-surface of the liver. This latter mass invades the substance of the liver and penetrates through the diaphragm.

"Upon opening the chest, both pleural cavities are found to be free of fluid and adhesions, save at the right base. The heart is normal. The left lung presents the usual signs of a bronchopneumonia. The right lung is firmly fixed to the upper surface of the diaphragm. Upon incising the right lung, there is found in the lower lobe a fibrous cavernous growth containing gelatinous material, communicating above with one of the larger bronchi (into which it has ulcerated). This is a direct extension of the process that had penetrated the liver and diaphragm.

"Specimens of this mucoid material were preserved and later examination showed it to be a pseudomucin like that found in the peritoneal cavity and that which had been expectorated a few weeks before death.

"Microscopic sections of the tumor mass show an alveolar type of growth rather than the papillary growth noted at the time of removal of the left ovarian tumor. This distinction, however, is not considered fundamental. The lining epithelium of these alveoli is several layers thick and irregularly flattened so that the individual lining cells are somewhat polyhedral.

SUMMARY

We have attempted to give a brief but fairly definite picture of the disease designated as pseudomyxoma peritonei and have presented a case showing many usual and some very unusual features. Among the former were the usual rapid enlargement of the abdomen, the initial pain thought to be pleurisy but probably due to rupture of the cyst, the questionable fluid wave bearing more resemblance to a thrill, the development of fever and peritonitis probably as the result of invasion of the intestinal tract, and the rather prompt recurrence of the disease.

The unusual features included the youth of the patient, her unmarried status, the presence of skin implants, the discharge of gelatinous material in the stool and most interesting, the extension of the process through the diaphragm with the subsequent establishment of a bronchial fistula.

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DISCUSSION

Dr. John W. Price, Jr., Louisville, Ky.: Dr. Hancock's case is of interest primarily because of the extension of the disease through the diaphragm and into the lung and externally. Proliferated papillomatous

cysts of the ovary are not unusual. They represent about 4 per cent of all tumors or growths of the ovary, and are frequently associated with the proliferated serous cysts which develop in the same ovary. About 20 per cent of these patients with proliferated papillomatous cysts show the growth to be present in both ovaries. The tumor, I think, is probably the most disastrous of all designated benign tumors, because of its tendency to rupture, to have transplants, and to produce adhesions and disability in the patients. Since the public has developed more confidence in the hospitals and in the medical profession as a whole we do not see many cases come to the hospital in a ruptured condition. As a rule, the diagnosis is made after the abdomen is opened and the specimen is removed.

I recall some twenty-two or twenty-three years ago having been called by Dr. Hancock's father to operate on a patient of his, with a ruptured papillomatous cyst of the ovary. That patient presented the typical symptoms. Since that time I have occasionally found these patients coming to the hospital with a ruptured cyst, but I do not recall ever having had any case where the growth extended beyond the peritoneal cavity.

Dr. M. J. Henry, Louisville, Ky.: Dr. Hancock has brought out an important factor in this condition, when he says it may be of very long duration. To emphasize that fact I wish to mention a case I operated upon in July, 1924, in which I found one of these cysts which had ruptured, a similar cyst in the other ovary, and a secondary growth in the appendix, all of which were shown microscopically to be malignant. The last report I had of that patient was about two months ago, when the doctor who referred her said that she was still working on the farm. That was over ten years ago and both ovaries, the uterus and appendix were said to have malignant mucinous material in them, or attached to them.

SOME OBSERVATIONS ON THE TREATMENT AND AFTER-CARE OF

FRACTURED BONES

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CHATTANOOGA, TENN.

WE have treated at the Newell Clinic during the past fourteen years, ending December 31, 1933, 5535 fractures, divided as follows:

TABLE I

1	Chest	471
2	Elbow	154
3	Femur	163
4	Tibia and fibula	570
5	Foot	1050
6	Radius	585
7	Ulna	347
8	Hand	1094
9	Head	355
10	Humerus	118
11	Knee (including 33 fractured patella)	132
12	Pelvis	182
13	Scapula	47
14	Clavicle	147
15	Spine	120
		5535

Open operations where not compounded were done on the following bones:

TABLE II

1	Clavicle	2
2	Humerus	4
3	Elbow	7
4	Femur	6
5	Radius and ulna	26
6	Tibia and fibula	15
7	Patella	26
8	Maxilla	7
9	Depressed zygomatic arch	9
10	Ribs	1
		103

All of the fractures were confirmed by *x*-rays. A record was kept of every fracture and a follow-up notation made on the records, as these were all private cases. Whenever any manipulation was necessary to correct deformity, a second, third or fourth *x*-ray was taken to determine the position of fragments after manipulation, traction or application of pressure pads. In fracture of important bones, a final *x*-ray was taken to determine the amount and density of callus before removing all supporting measures and definitely discharging the patient. We are more and more convinced that these terminal *x*-rays are very valuable and so often save the patient and the doctor from much chagrin and disappointment, as we have found that a union that seems firm by manipulation has not enough bony callus to justify excessive use and full weight-bearing.

We are having in the United States about one million fractures a year from auto accidents and since these fractures occur widely over the country, it is important that our doctors became familiar with satisfactory methods of treating fractures. The Committee on the Treatment of Fractures of the American College of Surgeons has been working as a committee for twelve years. They have done some good work but there is far more to be done as the treatment of fractures generally is still tragic in its results. Personally, I believe that the Committee could accomplish the greatest improvement in the shortest time by persuading the Medical Schools to better prepare their graduates for fracture work by giving more clinical demonstrations; then the young practitioner in the rural sections and small towns will educate and stimulate the older men.

There is not and should not be any standardization of the treatment of fractures, but there are some qualities that every man who treats fractures should possess and without which he will have many failures and disappointments. He must have the love for this work and everlasting patience, he must be familiar with the laws of traction and countertraction, have

FIG. 1. FIG. 2. FIG. 3.

FIGS. 1–3. Case 1. Fig. 1. Compound, comminuted fracture of middle third of left femur. This patient had to be treated for profound shock before any manipulation was done on fractured bones. Fig. 2. A. P. Free piece of bone seen in this picture is one-half of a two-inch transverse section of femur. Other one-half was removed from fender of car at time accident occurred. Fig. 3. Over-reduction from slight skeletal traction applied to malleoli.

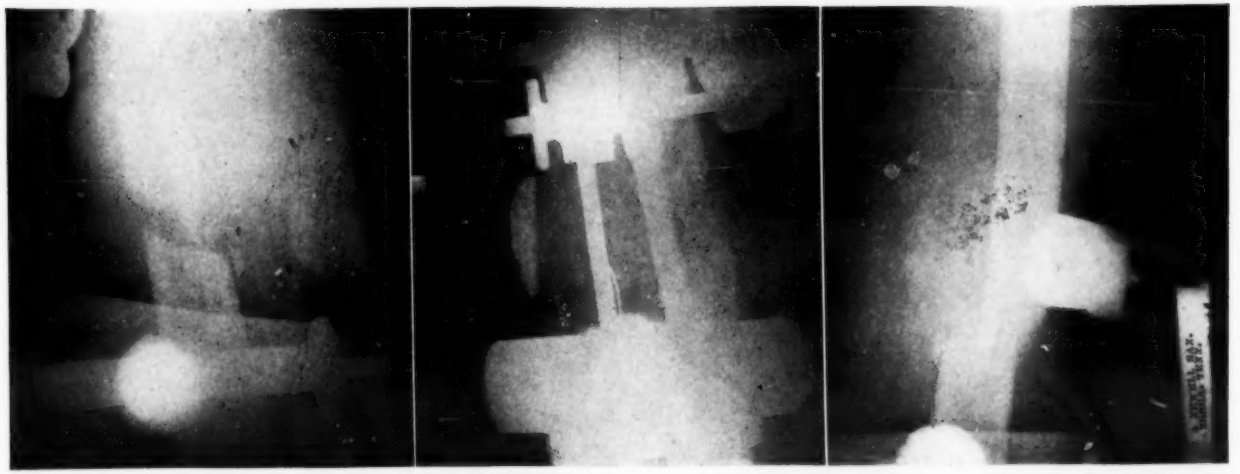

FIG. 4. FIG. 5. FIG. 6.

FIGS. 4–6. Case 1. Fig. 4. Apposition after traction has been lessened. Counter-pressure pads are seen holding ends of bones in apposition. Fig. 5. Later, showing better apposition. Fig. 6. Callus formation and piece of bone 2 inches long has been removed under local anesthesia as it was causing irritation.

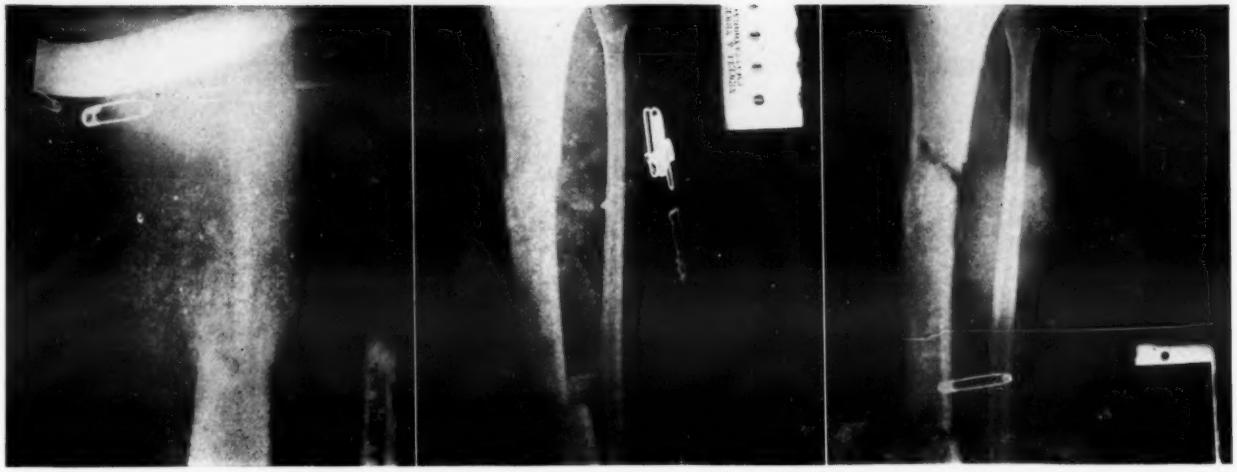

FIG. 7. FIG. 8. FIG. 9.

FIGS. 7–9. Case 1. Fig. 7. A. P. Fig. 8. Compound fracture in both proximal and distal thirds of tibia of same leg as fractured femur. Fig. 9. Same fracture after reduction with skeletal traction applied to malleoli, with just enough traction to keep bones in alignment and to prevent too much separation of fractured femur ends.

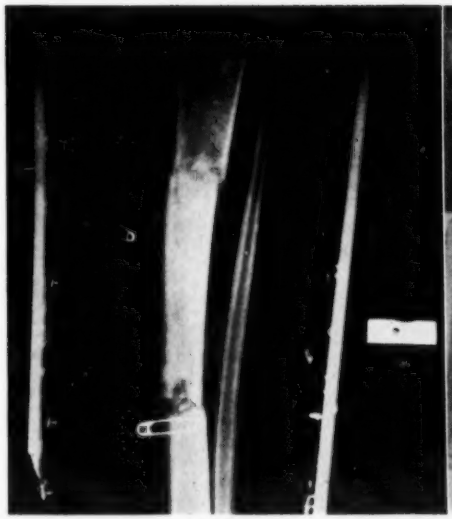

FIG. 10.

FIG. 11.

FIG. 12.

FIGS. 10 and 11. Case I. Fig. 10. Final results. Fig. 11. Final results, six months after date of injury. This patient walks with just a slight limp and has 50 per cent easy motion in knee-joint. Although exactly 2 inches of femur was removed, there is just one inch shortening of injured limb.

FIG. 12. Case II. Fracture of mandible near ramus on one side and compound fracture of mandible on other side opposite bicuspid. This same patient had multiple compound comminuted fractures of humerus near shoulder-joint, fracture of elbow-joint and fracture of both radius and ulna.

FIG. 13. FIG. 14. FIG. 15.

FIG. 13. Case II. Patient with loss of left arm at shoulder but otherwise in good health. He has returned to duty. All sinuses of face have healed and there is firm union with good apposition of both fractures of mandible. This patient was in such profound shock when first admitted that he had to be given both glucose intravenously and blood transfusions. Mangled arm was cleansed with soap and water and ether and covered with sterile gauze dressing. No manipulation was done and no attempt was made to wire fractured mandible at this time. In three days, under local and gas anesthesia, arm was amputated at shoulder and fractured mandible wired.

FIGS. 14 and 15. Case III. Badly compound comminuted fracture of both tibia and fibula with marked trauma and destruction of soft parts. It seemed impossible to save his foot but due to pleadings of patient we enlarged traumatic opening and placed many fragments of bone together and then closed wound without drainage. Much to our surprise this patient did not have infection. He developed good bony union and was back at work ten months after injury as switchman in railroad yard, with no other support than an Unna paste boot. Fig. 15. Patient without any shortening and with practically no deformity of his leg.

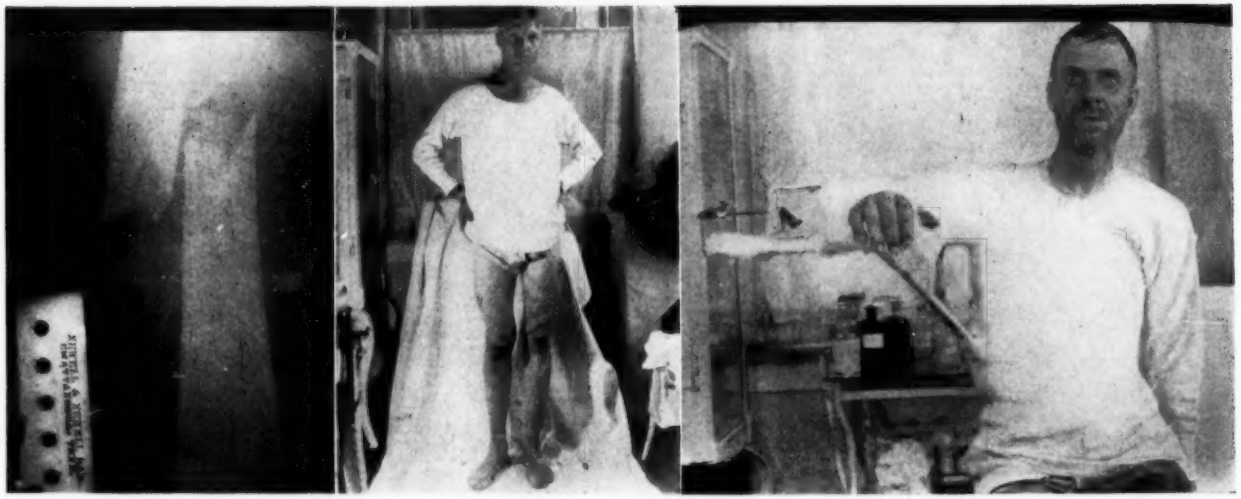

FIG. 16. FIG. 17. FIG. 18.

FIGS. 16 and 17. Case IV. Compound fracture of right femur in upper one-third with proximal bone protruding through an opening in skin. Patient in profound shock. It was necessary to give two blood transfusions before any traction could be applied or any manipulation done other than to apply a Thomas splint to prevent further shock. Fig. 17. Patient six months after injury. There is only a slight shortening of injured limb. This case was treated by skeletal traction applied to condyles of femur.

FIG. 18. Case V. Our method of treating fractures of humerus. This patient was not willing to stay in hospital so plaster cast was applied over a heavy cotton shirt without any other bandage. Triangular wooden frame was made in office by our orderly and traction was applied by applying moleskin adhesive plaster to lower end of humerus and placing usual wooden block between two adhesive strips. A cord was applied to an upright piece which was screwed to wooden frame and sufficient traction applied until bones were shown under fluoroscope to be in perfect apposition. This splint can be applied by any doctor who has plaster of Paris, a few pieces of board, and who has a little ingenuity and enough interest in his work. We find this form of splint more satisfactory than any of the expensive splints on the market.

some mechanical skill, be ever watchful for the patient's comfort and for the proper fitting of splints and appliances, and not to know the anatomy of the fractured part or the

FIG. 19. Case VI. Volkmann's ischemia, showing inability to extend fingers.

pathology of fractures will mean disaster early and often. With these qualifications splendid fracture work can be done in isolated areas without hospital care and without the vast unnecessary paraphernalia that clutters up the stockroom of so many hospitals. This fall while dove shooting with some city sportsmen, wearing the most approved hunting coats and caps and shooting automatic Brownings, Parkers, and Winchester pump guns and using Dupont smokeless powder, I observed that their marksmanship was indifferent. In the middle of the field close to me, I noticed a gangling lad of fourteen, wearing cotton trousers, a flannel shirt, bareheaded and barefooted, shooting a pot-metal single barrel, breech-loading gun. He killed practically every bird that came his way and he was shooting the cheapest black powder shells that are sold. He had the instinct, he had inherited the spark! I have written many papers on fractures and I have changed my opinion about some particular feature of treatment just as often, but there are some dogmas I formulated that I have not changed.

A fracture of a long bone is an emergency and the treatment should begin as soon as the fracture occurs; no rest hour or meal hour should interfere with the immediate treatment of that fracture. The reduction, if possible, should be done with the very least amount of manipulation and trauma and when

reduction is satisfactory, immobilize and keep it immobilized until union is complete. If satisfactory reduction cannot be completed at once by manipulation and traction, endeavor to

FIG. 20. Case VI. Volkmann's ischemia, showing inability to extend fingers.

terminate your efforts at satisfactory reduction at the end of ten days. After ten to fourteen days, I think it is very risky to further endeavor to change the position of the broken ends. Where manipulation is necessary, we use gas or ether anesthesia, or local anesthesia, with preference for ether anesthesia if much relaxation is required. We use skin or skeletal traction as is indicated for each particular case, but always skeletal traction if much traction is required. We use pins, wire, or various types of ice-tongs indifferently, as will be best adapted for each particular case.

After satisfactory reduction and immobilization with circular plaster cast, molded plaster splints, board splints, Thomas splints, etc., we then begin encouragement for slight active movement of the muscles of the broken limb, gradually increasing the muscular movements to increase the circulation of the limb, thus stimulating callus formation and later osteogenesis and preventing great muscular atrophy from dysfunction.

When this is carried out from the beginning and you have the full cooperation of the patient, which is so necessary in the treatment of fractures, when the time comes to remove the

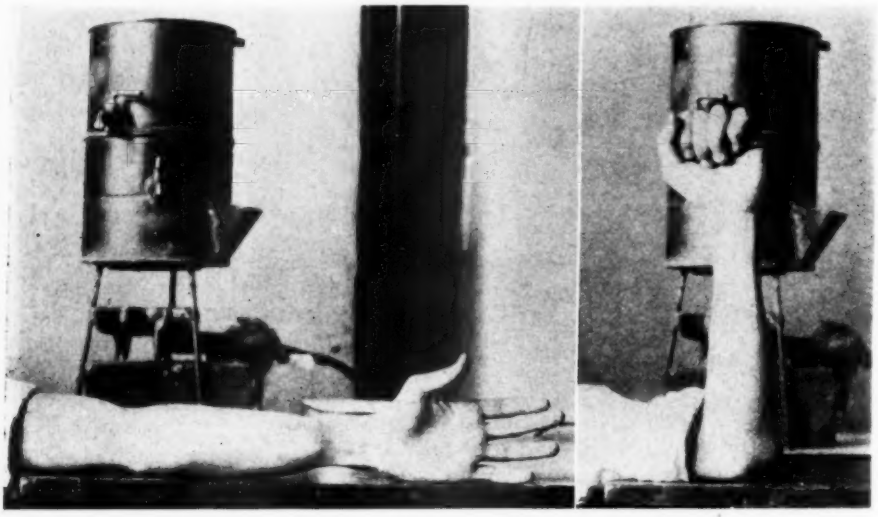

FIG. 21. FIG. 22.

FIGS. 21 and 22. Case VI. Final results, eight years later.

splints, there will be no necessity for the very expensive and more than useless hours, days and months spent in the physiotherapy rooms with masseurs, bakings, radiant lights, diathermy, hot and cold plunges ad absurdum. It is bad for the patient because it prevents him from doing something for himself that others cannot do for him. I show my patients what they can and must do by active motion, believing that ten minutes of active motion is worth hours of passive motion and manipulation. All patients are encouraged to return to work early, to work they are accustomed to or as much of the work as they can do. If not employed, then they are made to play at such exercises as will increase motion in joints and stimulate muscle contraction. If you can inspire the full cooperation of your patient, he will work for you to regain quickly the full normal use of a broken limb and physiotherapy will not be necessary.

In the series of 5535 fractures we have had 4 cases of non-union: non-union of the radius occurred once, of the humerus twice and of the tibia once, all in healthy, robust, white males. We have never had non-union in a negro patient.

Interposition of muscles or tendons between the broken bones has never been the cause of non-union in any of our cases.

Early satisfactory apposition and continued immobilization are the two essential factors to obtain early, firm union of broken bones and early active motion is the essential factor to obtain full normal function of the injured limb.

SUMMARY

We have treated at the Newell Clinic during the past fourteen years, ending December 31, 1933, 5535 fractures divided as shown in Table 1.

Open operations where not compounded were done on the bones listed in Table 11.

In the series of 5535 fractures there were 4 cases of non-union: radius 1; humerus 2; tibia 1.

[For Discussion see p. 765.]

PROBLEMS IN THE TREATMENT OF

FRACTURES OF THE LONG BONES*

EDGAR LORRINGTON GILCREEST, M.D., F.A.C.S.

SAN FRANCISCO

MOST fractures, particularly those of the long bones with comminution or displacement of fragments, should be regarded as potential deformities. In an effort to prevent, or at least minimize, such deformities, every fracture should be studied carefully as to the best procedure to follow in order to secure restoration of length and, if possible, anatomical reposition of the fragments and early return of maximal function. Each fracture presents its own problem and often taxes the surgeon's ingenuity, judgment and patience.

I believe that primary operative reduction of fractures will never become the recognized or accepted routine of treatment. Our chief attention, therefore, should be directed to perfecting non-operative methods. In fractures of the long bones with displaced and overriding fragments, skeletal traction-suspension is the most reliable means of treatment if immediate manipulation fails to accomplish proper reduction. In a majority of cases this traction skillfully applied, carefully maintained, and aided by lateral or anteroposterior pressure-pads of proper size and shape, as indicated, will give satisfactory and often perfect anatomical as well as functional end-results.

The continued use of adhesive plaster in an attempt to secure restoration of length in fractures of the long bones in adults explains many poor results still seen today. Traction by adhesive plaster requires double the weight needed by skeletal traction to exert the same pull on the bone and, therefore, often will not withstand an adequate pull. In addition the skin may become irritated and blisters form. In chil-

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dren, however, adhesive plaster, or, preferably, moleskin plaster, has a definite use in the treatment of fractures of the long bones in which one would hesitate to insert a wire near

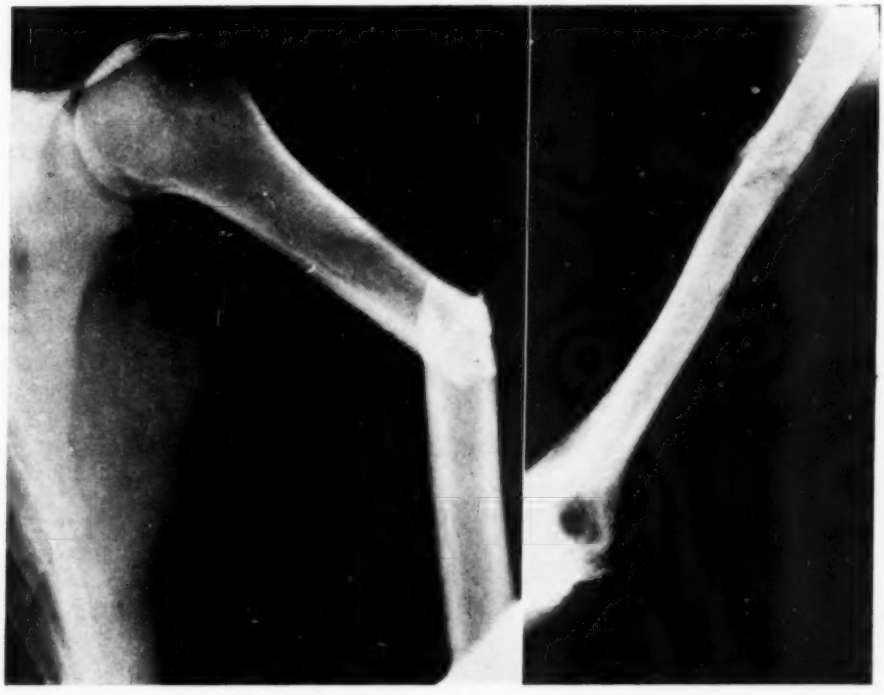

FIG. 1. FIG. 2.

FIG. 1. Case 1. Transverse fracture of left humerus at junction of upper and middle thirds with over-riding and angulation.

FIG. 2. Case 1. View of humerus four months later showing perfect alignment secured by traction with Kirschner wire and Hoke's plaster cast.

the epiphyseal line for fear of interrupting the growth of the bone.

In the application of skeletal traction one should consider the best means to secure this by the least trauma to the skin, underlying tissues and bone. The non-penetrating caliper, a modification of the Schmerz clamp and Ransohoff's ice tongs, often has served satisfactorily. Its chief disadvantages are the necessary size of the points about which irritation, pain and infection ensue, aggravated by the rotatory movement which invariably occurs about the point of grip. Often the exact size

of caliper required for the particular case is not available at the time it is needed. Further, any caliper, because of the technical difficulty of insertion, frequently pulls out of the

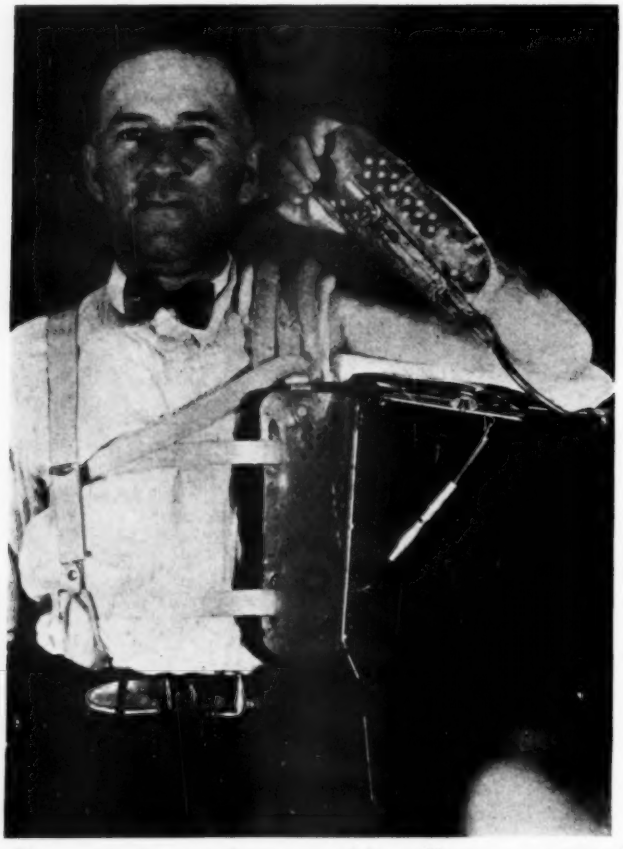

FIG. 3. Case 1. How a contracture from wound in axilla was overcome by abduction splint with a turnbuckle which permitted gradual abduction of arm from body. Recently united fracture of humerus was adequately supported and by means of a special hinge at elbow forearm could be flexed or extended and arm internally or externally rotated, as desired, thus gradually regaining range of motion which had become restricted.

bone, if not properly placed, and irritates the soft parts.

The Steinmann pin (Fig. 12) with Finichetti stirrup, especially if made out of rustless steel and of suitable size, is better "as it remains quite firm; it does not turn nor rotate in the wound; it will not slip nor pull out; it makes a uniform

pull on the bone at a right angle to the long axis of the limb; and it is very dependable for embedding in plaster of Paris." (Kellogg Speed.)

FIG. 4. FIG. 5.

FIG. 4. Case 11. Anteroposterior view of left femur in diabetic man seventy years old. Note severe comminution and displacement.

FIG. 5. Case 11. Lateral view. Note classical posterior displacement of distal fragment and also three large loose fragments.

The Kirschner wire has the advantage over the Steinmann pin of being much smaller in caliber and, therefore, of producing less trauma and irritation. This fine stainless steel wire, which easily can be passed through the bone where desired, is pulled readily and held taut by the traction clamp. The satisfactory results obtained by this wire account for its increasing popularity. (Figs. 1, 2, 4–9.)

The sooner traction is applied the more easily is overriding corrected and alignment secured. Every hour of delay means increased muscular spasm, which requires additional weight

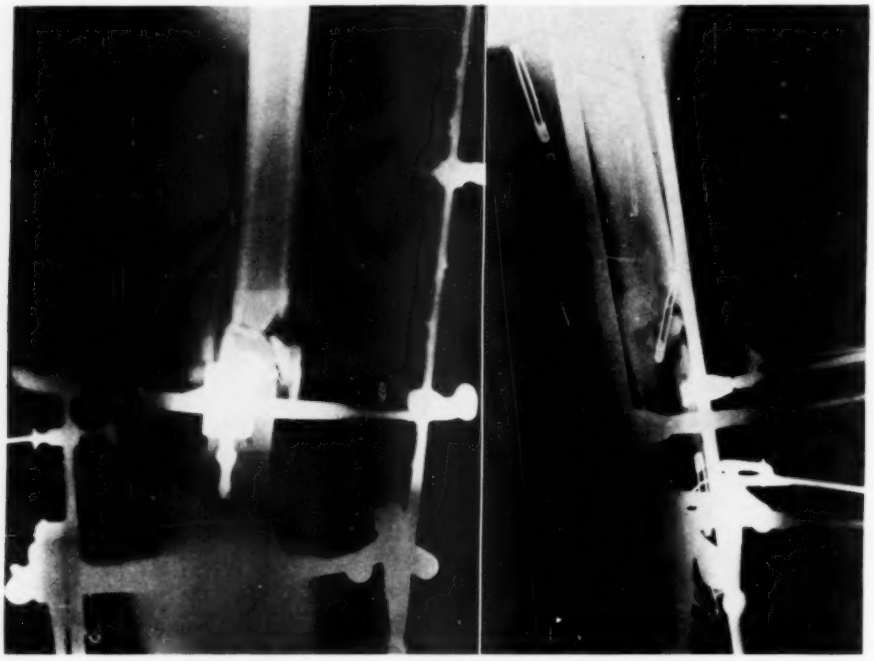

FIG. 6. FIG. 7.

FIG. 6. Case II. Immediate perfect alignment secured under local anesthesia by means of traction-suspension through Kirschner wire and Thomas splint. Alignment was maintained by means of pressure pads.

FIG. 7. Case II. Lateral view showing anterior pressure pad preventing distal fragment from displacing forward after wire had been inserted.

to overcome and in turn necessitates an undue strain on the ligaments and muscles. The immediate application of traction at the site of injury, as carried out during the latter part of the World War and later emphasized by Kennedy, is very important. No measures will lessen pain and decrease swelling so quickly as early reduction. It is the height of absurdity to wait for swelling to subside before attempting reduction. Every fracture, therefore, should be considered as an emergency, as every hour's delay not only may mean additional

time for convalescence but may increase the probability of partial permanent disability.

Skeletal traction once instituted should have sufficient pull

FIG. 8. FIG. 9.

FIG. 8. Case 11. Note perfect anatomical alignment.
FIG. 9. Note perfect anatomical alignment. End result: splendid function.

in the beginning to overcome the overriding. Most surgeons make the error of adding too little weight at first rather than too much, thereby requiring more weight a few days later than would have been necessary. With adequate traction restoration of length should be obtained in twelve hours or less. One should not hesitate to apply to a fracture of the femur from 25 to 30 pounds' traction, at the very beginning of treatment. Until satisfactory apposition and alignment are secured a knowledge of the position of the fragments should be obtained

by a daily roentgenological examination. Failure to follow this rule often leads to serious results. With reduction once obtained the weight should be reduced gradually.

FIG. 10. FIG. 11.

FIG. 10. Case IV. Anteroposterior view of severely comminuted fracture of lower third of left tibia and fibula involving joint. Note marked separation of fragments and severe inversion of foot.

FIG. 11. Case IV. Lateral view showing great displacement of distal fragments.

Our skill in the use of various methods of traction should not make us forget that we still have hands and that our predecessors generations ago used theirs, with at least reasonable success, both for the diagnosis and the treatment of fractures. Apparatus, therefore, should supplement, not usurp, manual reduction. Every age has produced its "bone-setters" who developed remarkably delicate tactile sensation in their

fingers and great manual skill and dexterity, as evidenced by their successful reduction of fractures. Seen early, many fractures can and should be reduced manually under either

FIG. 12. FIG. 13.

FIG. 12. Case IV. Anteroposterior view. Note that immediate perfect alignment was secured.

FIG. 13. Case IV. Lateral view. Note also perfect alignment of both bones.

local or general anesthesia. The ease by which manual reduction is accomplished is commensurate with the time which has elapsed since the fracture occurred. This is particularly true in fractures of the elbow, wrist and ankle where excellent reductions are often obtained by this method. For example, if a Colles' fracture is seen a short time after it occurs, it is often possible to secure a perfect reduction by careful manual manipulation without the use of anesthesia.

In a certain number of fractures there will be non-union, caused by various factors beyond our control, such as the interposition of tissue between the fragments. These cases

FIG. 14. Case IV. Anteroposterior view showing sliding autogenous inlay graft but no union after several months.

require surgical intervention such as drilling and inlays and onlays of autogenous grafts, followed by immobilization in splints or plaster. The procedure to be followed should be considered carefully. Often a simple method will stimulate osteogenesis and then the use of a cast with a walking stirrup for fractures of the leg will bring about a firm union. Figures 10–13 illustrate how a severely comminuted fracture of the lower third of the left tibia and fibula was corrected and a satisfactory alignment obtained. Union, nevertheless, did not occur in the tibia and an autogenous, sliding, inlay graft was

used. Union still failed to occur (Fig. 15) until weight bearing by means of a walking stirrup in a cast was used. Within a few weeks abundant callus was produced and a splendid end-

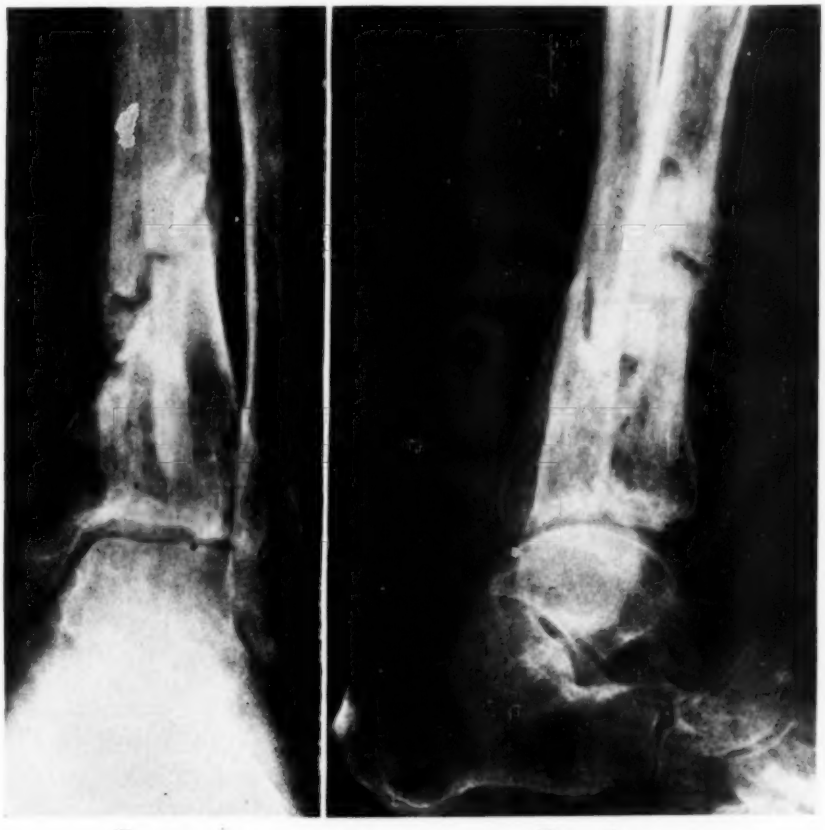

FIG. 15. FIG. 16.

FIG. 15. Case IV. Anteroposterior view showing perfect alignment and firm union.
FIG. 16. Case IV. Anteroposterior view showing perfect alignment.

result was obtained. (Figs. 15 and 16.)

One should not focus one's entire attention on the fracture and neglect the injury to the soft tissues. Swelling, which always ensues, interferes with the vascular and lymphatic circulation of the part. This can be reduced greatly by elevating the extremity and by beginning massage immediately. Early and carefully guided active and passive movements of the joints should be carried out. When union is sufficiently firm,

various splints should be used in order to make the patient ambulatory as soon as possible.

SUMMARY

1. All fractures should be regarded as emergencies.

2. All fractures of the long bone should be viewed as potential deformities.

3. Traction splints should be applied, if possible, at the site of injury.

4. Primary operative reduction of fractures will never become the recognized or accepted routine of treatment.

5. Manual reduction under local or general anesthesia always should be considered first.

6. Traction by adhesive or moleskin plaster has only a very limited usefulness and accounts for many unsatisfactory results.

7. Skeletal traction-suspension, skillfully applied and carefully maintained, usually gives excellent results. Surgeons, nurses and hospital attendants should familiarize themselves with this method.

8. In cases of non-union or delayed union, drilling, inlay or onlay grafts may be necessary.

9. Massage should be instituted at once and active movements of joints begun early. With their fractures properly protected all patients should be made ambulatory as soon as possible.

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DISCUSSION OF PAPERS OF DRS. NEWELL AND GILCREEST

DR. GEORGE E. BENNETT, Baltimore, Md.: In treating fractures conservatively, I think if we would just use a little ingenuity when applying apparatus of one sort or another we would be very apt to secure satisfactory end results.

I was interested in Dr. Newell's case of non-union of the small bones where union usually occurs. Of course, I doubt if the results obtained in fracture cases on a regular hospital service would compare favorably with those of private patients, where individual attention to detail is given by the surgeon in charge. I think our percentage of non-union runs higher than Dr. Newell's but my own percentage of non-union is relatively small.

There is one point that I should like to mention here and that is the use of the fluoroscope. I believe that we, particularly those of us in teaching positions, should discourage the too frequent use of the fluoroscope because the men will learn to depend too much on it and thereby lose the art of reduction. We should learn to use our hands and not rely on the fluoroscope alone. I know we are all prone to fall into bad habits and I have found, if

permitted, men reduce every fracture under the fluoroscope and then when they leave the hospital and attempt to reduce a fracture by manipulation they are not capable of doing it.

DR. FREDERIC W. BANCROFT, New York, N. Y.: I should like to bring to your attention what the Committee on Fractures of the American College of Surgeons is attempting to do toward educating the public in immediate traction for fractures along automobile highways. The harm that is done by the jackknifing of these fractures in transportation is known to all of us.

The regional committees in New York, Chicago and Boston have found that in ordinary ambulance transportation the Thomas splint for the lower extremity and the Murray-Jones splint for the upper extremity were not utilized by the ambulance. In each of these cities, at the instigation of the local committees, these splints have been supplied and the ambulance aides have been taught the method of application. Before this time the doughboy in France had a better chance, after a fracture, than did the ordinary civilian in any large city. The problem of having splints supplied to ambulances is a difficult one. A large percentage of ambulances are run by undertakers, police and firemen. I think it is up to each of us to see that the ambulances in our particular city are equipped with Thomas splints and that the ambulance corps are taught the method of application.

In the Lincoln Hospital in Chicago (a hospital run by colored doctors and a postgraduate center for negro doctors in Illinois) they have been able to make Thomas splints for $1.00. I think any of us could get a blacksmith to make these splints at a very reasonable price.

The American Red Cross is desirous of having first-aid stations, equipped with Thomas splints, along main highways. At the present time their lack of sufficient finances has prevented this being done. However, in Arizona, under the local regional fracture group, the state police have been taught the application of immediate traction and we hope before long the highways there will be well equipped. I believe each of us can aid in this campaign of education in his own community.

DR. JOHN E. CANNADAY, Charleston, W. Va.: I wish to congratulate the two essayists on their results, and on their improvization of their own apparatus. In looking over a number of the hospitals in this country I find it stressed in several of them that apparatus must be made by their own personnel. I understand that in the Cincinnati General Hospital all the fracture apparatus is made by the resident staff, under proper direction and control, of course, and that is the way it should be. Our young men in training should be taught self reliance. They should not feel that everything has to be bought.

I want to stress the importance in all fractures of securing anatomical restoration if possible. If not absolutely necessary, it is highly desirable. In the fractures of the long bones the principles of traction, countertraction and fixation are exceedingly important. It may always not be so great a job to reduce a fracture but it may be a considerable job to maintain reduction.

I think what Dr. Bennett said is to be emphasized. We should be able to reduce fractures without using the fluoroscope, but the fluoroscope is a nice thing to use to check up to see if the fracture has been properly reduced.

DR. E. DUNBAR NEWELL (*Closing*): I agree as to the use of the fluoroscope. First I take an *x*-ray picture, then I manipulate and take another picture to see if I am correct. Then I file it away for legal reasons.

In reference to Dr. Bancroft's remarks, we now have every ambulance fitted with a Thomas splint, and the attendants have all been instructed that every fractured limb must be immobilized and brought to the hospital at once, and nothing done to it besides this. I think this is the most important work the Fracture Committee has done.

I think it remarkable that Dr. Gilcreest and I, without any previous discussion, have arrived at the same point—the antagonism against open operation. If you use enough intelligence and enough diligence you can get along without open operation in 90 per cent of fractures. While we have had a few open operations and have as yet had no infection, I feel that by doing the work in the simplest manner possible we get the best results. We have found that in all open operations the return of firm union has been extended at least 50 per cent, even in the cases where there is no sign of infection. That is the reason we always strive to avoid them.

DR. EDGAR L. GILCREEST (*Closing*): Dr. Newell has emphasized some very important and essentially practical points in the general management of fractures from the time they first occur. It is this early care coupled with Dr. Newell's rare judgment in the choice of non-operative procedures which has made it possible for him to show us such splendid end-results. All civil ambulance drivers today should be taught how to apply the Thomas splint just as the drivers of ambulances were taught during the war.

I believe that in the transportation to the hospital of fracture cases and then in moving them from bed to guerney for roentgenograms much trauma occurs to the soft tissues in and about the fractured area. If splints are applied at the site of the accident and patients are taken directly to the *x*-ray room before being put to bed, less displacement will occur, the patient will suffer less, and there will be considerably less muscular spasm to be overcome. Restoration of length and alignment, therefore, will be obtained earlier.

MALIGNANT CHANGES IN THE SO-CALLED BENIGN GIANT-CELL TUMOR

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THE question of whether giant-cell tumors of the long bones are always benign in character and, therefore, should be taken out of the class of *neoplasms* or whether a certain and quite appreciable number later take on malignant features and cause death from metastases, is a very old one, and has been the subject of much debate for over a hundred years. Sir Astley Cooper in his "Surgical Essays" (1818)[1] gave a very accurate description of the giant-cell tumor, noting the expansion of bone, increase in swelling, ulceration, sloughing and bloody discharge, and stated:

> The fungus projects considerably, the skin yields very extensively, and so at length slough takes place by means of which considerable portions of the swelling are separated and the disease becomes so far diminished in volume as to induce a hope of its ultimate destruction by gangrene; a hope, however, which in this complaint of the bone, I have never seen realized. . . . The time occupied in the destruction by these means is sometimes two years and at others is a much more protracted period as from seven to ten years. It often happens in this disease that tumours of a similar kind form in other parts of the body during its progress; so also where the affected limb has been amputated, a similar disease will occur at a future period and in the organs of greatest importance.

This was followed in 1845 by a fuller description by Lebert,[2] who is generally credited as being the first to express the belief that giant-cell tumors are always benign. Sir James Paget,[3] nine years later, gave a still more accurate description. He also called attention to the benign character of these tumors but he very wisely refrained from any dogmatic statement, and qualified his opinion as follows: "Still, I suppose, cases may be found in which, with the same apparent structure, a malignant course is run."

Nélaton's[4] classical monograph of 355 pages was published in 1860. While no one hesitates to give him full credit for his splendid report on giant-cell tumors, its real value lies in his careful clinical and histological studies and in the very fine drawings of both the gross and microscopic sections of giant-cell tumors. Curiously enough, Nélaton is quoted as being among the earliest and most important authorities for the opinion that giant-cell tumors are always benign and never metastasize. This is possibly due to the fact that his book was published seventy-four years ago, and that it had a very limited circulation, only a few copies being available for reference in this country. I believe that a careful study of Nélaton's original book will show it to be an error to regard him as an authority on the prognosis of giant-cell tumors. An analysis of the series forming the basis of his study shows that it was limited to 46 cases, mostly collected from medical literature, and that only 6 cases came under his own personal observation. Of the 46 cases, the great majority were epulis of the jaw, and only 14 were giant-cell tumors of the long bones. Of these 14 cases, all were treated by amputation. As these cases occurred before the days of Lister, and when a follow-up system was unknown, it is not surprising to find that 4 died of infection, only 2 were traced alive and well two years later, 4 were well one year later, and the rest were not traced.

Only two years after the publication of Nélaton's book, Virchow,[5] the greatest pathologist of the century, brought out his admirable work entitled "Die krankhaften Geschwülste." In suggesting how giant-cell tumors may recur and may even prove malignant, he assumes a progressive infection, and states that this explains why after superficial and limited removal of tumors, local recurrences in bones or soft parts are so frequently seen; while removal with extensive resection or exarticulation is followed by safe and lasting cure for a certain period of time. He adds that the local malignancy of epulis is well known. Hence, the older authors since Paré and Fernel speak of a "degeneration into cancer."

It is interesting to note that Virchow, even in that day, recognized the marked clinical difference between giant-cell tumors of the jaw (epulis) and myeloid sarcoma of the long bones. Of the epulides, he states that except for their local malignancy, they have never given cause for suspicion; however, of the myeloids of the long bones, a number of undoubted cases of highly malignant metastases are known. It is true that later on these cases were considered as complicated and the metastases as cancerous, but, in Virchow's opinion, wrongly so. An older case of Gerlach's, described by himself as cancer, may be doubtful as to its point of origin:

A youth of seventeen years died of a cortical bone tumor of the lower end of the femur containing a large number of cells in its outer soft parts, among them "many fairly large ones with endogenous nuclear formation (mother cells)." In the lungs and mediastinum were metastatic soft nodules in which the large mother cells and cell nuclei were very numerous, while the "fibrous structure was crowded back."

That this was not a cancer, Virchow states, but a sarcoma with myeloid cells in the present-day sense, is clear.

Again, Virchow cites the case of Cooper Forster:

A male, aged eighteen years, with a tumor the size of two fists, in the fibula. The original tumor contained, outside of disseminated bone parts and cysts with blood and serum, an encephaloid mass, with sections here and there showing cells of "truly myeloid character." A recurrence took place three months later, and death from metastases in the spine and lung, two months later. Wilks pronounced the metastatic tumor as similar to that of the fibula; he found bone tissue, nuclei, nuclear cells and some myeloid cells; and he reported the case as a combination of osteoid cancer with myeloid.

Virchow mentions still another case, that of Mitchell Henry:

A female, aged forty-three years, had complained of pain in the right shoulder for twelve months; ten months later, on exarticulation, a tumor the size of an orange was found in the head of the ulna, partly bony and partly fibrous. In the medullary cavity was a soft, reddish mass which extended into the tumor, and consisted almost entirely of myeloid cells plus a granular mass with nuclei and spindle cells. Eleven weeks after the

operation, a recurrence took place in the stump. Death followed in a short time. Autopsy showed a tumor of the scapula, the size of a child's head, involving the axillary glands, consisting of porous osseous lobules, separated by fibrous bands and blood cysts; the lungs contained from twenty to thirty nodules, the size of a pea. The bony part of the scapular tumor showed bony corpuscles without canulae, the soft part showed small cells and nuclei, large cells with one or more nuclei, fibroplastic cells and almost everywhere myeloid cells with from three to fourteen nuclei. The nodules in the lung contained but few fibrous parts, but distinct bone tissue and less numerous myeloid cells.

Virchow finally cites a case by Cook and Wilks, that practically excludes all doubt:

A male, aged thirty-three years, was operated upon (amputation above the knee) for a scaly myeloid of the head of the fibula, first noticed seven months previously. After satisfactory wound-healing, the man resumed his work. Two years later he returned with a recurrence. A few smaller and one large nodule were extirpated. They proved to be scaly myeloids, not connected with the bone. A few days later the patient died of pleuritis. Three or four nodules were found in each lung; they proved to be myeloid; there was no glandular involvement.

After these experiences, Virchow believes there is no longer any justification for calling myeloid tumors benign. The objection made, that these tumors were not pure myeloids, he does not understand, inasmuch as there is no such thing as a pure myeloid; they are always mixed; and the doubts expressed regarding the reported cases would hold good with equal justice in practically all other myeloids, and particularly the cured cases. The fact is, that also the multinuclear giant cells occur in metastatic nodes, e.g., of the lungs, and therefore behave just as though they were cancerous elements.

Gross, in his classical paper on "Sarcoma of the Long Bones,"[6] took issue with Nélaton, and reported 5 cases of "benign giant-cell tumor" from the literature, in which metastases had occurred.

Passing over without mention these older cases of Virchow and Gross, Geschickter and Coperland[7] have selected for consideration 8 cases which they have collected from recent

literature and from the records of Johns Hopkins Hospital (a series of well over 500 cases) and which are supposedly the most convincing cases thus far available. These are the cases of Turner and Waugh, of Wilson, of Goforth, of Finch and Gleave, of Chatterton and Flagstad, of Dean Lewis, of Martin, Coley, Stone and Ewing, and of Dingman. They summarize as follows:

> We may conclude from an analysis of these eight cases selected as avowedly metastatic from among hundreds of typically benign giant cell tumors that they are not strictly bona fide. In no case has transformation of giant cell tumor into sarcoma been proved, and in no case have typical giant cell tumor nodules been found as metastases in the lungs. In half of the cases a diagnostic error was made, either in ascribing death from other causes erroneously to metastases, or in failing to recognize the histology of the original lesion as sarcoma. In the other four cases, since material from the original lesion was not saved for confirmation of the diagnosis, there is no proof that the original tumors were typical giant cell tumors, and not a low grade malignancy from the start, nor can it be contended that the giant cell areas in the final sarcomatous growths were other than a resorptive phase in osteogenic sarcoma without histologic connection with giant cell tumor. The only plausible deduction is that in a few isolated instances an apparently benign lesion of bone, when subjected to unsuccessful treatment and to trauma, may by its failure to heal, provide a fertile site for the subsequent development of osteogenic sarcoma. The unhealed area of bone, and not the nature of the original lesion is the important factor.

This, to me, does not seem to be a judicial or a final solution of the problem. If we accept it we must admit that all giant-cell tumors are benign and never metastasize. I, for one, cannot accept the statement that there are no authentic cases on record in which nodules in the lung have been found having the same histological structure as the primary giant-cell tumor. Earlier reported cases, apparently overlooked by Geschickter and Copeland, as well as newer evidence obtained from our own series, refute this.

Again, these authors assume that there are only one or two pathologists capable of making a correct diagnosis of

primary benign giant-cell tumor, since they exclude as an error in diagnosis the Memorial Hospital case (Case v) in spite of the fact that the primary tumor was examined and pronounced a benign giant-cell tumor by Ewing, Wolbach, Mallory, the entire Bone Sarcoma Registry committee, and finally, Bloodgood.

A study of this case was made by Stewart[8] of Leeds, and although he was formerly quite as convinced as are Geschickter and Copeland of the benign nature of giant-cell tumors, he regarded this as a true example of a benign giant-cell tumor that had become malignant. In a personal letter to me he stated:

> The practical conclusion is that there occur cases, clinically and pathologically indistinguishable from ordinary myeloid sarcoma, in which an unusually high grade of malignancy exists, including even the power of dissemination. Such cases are obviously of rare occurrence, but the possibility should be borne in mind by surgeons.

Unfortunately in the Memorial Hospital case there was no autopsy, but roentgenograms taken a few weeks before the patient's death showed both lungs filled with many typical metastases. Whether or not a histological examination of these nodules would have shown a typical giant-cell structure, the same as the primary tumor, it is impossible to say, but I regard this as a matter of little consequence. The main point is, that the patient died of metastatic tumors of the lungs. Furthermore, there are other cases that have apparently escaped the notice of Geschickter, Copeland, and Bloodgood in which a post-mortem examination has shown metastatic nodules of the same structure as the original giant-cell tumor.

Among these I would mention first the case of Auge and Roux.[9]

> This patient, a male, aged twenty-two years, had complained of pain for a period of two months following an injury. A pathologic fracture had developed one week later. An amputation was performed, and the patient died two months later of general metastases, including the lungs. Fortunately, a post-mortem examination was made. This showed the lung filled

with numerous hemorrhagic nodules or growths, together with commencing pericardial involvement; the left kidney was almost completely replaced by tumor. Microscopical examination of the primary tumor showed very many typical multinucleated giant cells, including many enormous forms with innumerable nuclei; these seemed to be most abundant where there was necrosis and hemorrhage, and occurred chiefly in the central part of the tumor. The mixed-cell ground-work was composed of cells which showed great variety both in size and shape; there were many mitotic figures. Nearly all of the nodules from the lungs and the secondary deposits in the kidneys showed numerous multinucleated giant cells (one tiny nodule in the lung showed no giant cells).

One of the most convincing examples of a metastasizing "benign" giant-cell tumor of bone is reported by Dyke[10] of Wolverhamptom, England. This author calls attention to the fact that while "opinion as to the malignancy or otherwise of that tumor of bone which used to be known as the *myeloid* sarcoma but which is now more generally spoken of in this country (England) as the *osteoclastoma* and in America as the *benign giant-cell tumour of bone* has varied a good deal in the past, of late there has been a tendency, reflected in the name in general use in America and under which it appears in the American Registrar of Bone Sarcomata, to regard it as essentially benign and incapable of metastasis." In the opinion of Dyke, his case would appear to call for a revision of this view.

Dyke's patient, a male, aged twenty-five years, had been well until July, 1924, when he injured his left knee while at work. This did not give him much trouble until the following October, when he consulted a doctor, who found some effusion into the joint but no marked disability. In November the fluid had disappeared and the leg was less swollen. The patient was able to carry on his work as a gardner until the end of 1925 when there was increased swelling and a return of the pain. On December 21, 1925, he was admitted to the Royal Hospital, Wolverhampton, with a provisional diagnosis of tuberculous disease of the knee-joint. A roentgenogram taken at this time showed the femur and tibia apparently normal, but the usual structure of the patella had disappeared, and there were "fluffy" areas of new bone formation, suggestive of new growth. An operation was performed by Mr. E. Deanesly, who found the patella to be enlarged and filled with reddish growth. The bone was curetted, and the wound closed; healing

took place by first intention. *Microscopical diagnosis:* giant-cell tumor of bone. During the next three years, the joint gave little trouble, and the patient was able to carry on his work. Towards the end of February, 1929, he slipped and hurt his knee which became swollen and hemorrhagic. The swelling increased rapidly and an amputation was advised by Dr. Spackman but refused. Towards the end of March he was given a series of injections of Coley's fluid but without effect. A disarticulation at the hip-joint was performed on April 28. Examination of the amputated leg showed a fixed swelling about the knee-joint. On cutting into this swelling it was found to consist of a mass of new growth occupying the site of the patella and invading the lower end of the femur. *Microscopical diagnosis:* typical osteoclastoma (giant-cell tumor). The patient remained well throughout the summer of 1929, but in October, 1929, a small nodule was noticed upon the scalp. This was removed and examined microscopically, and pronounced: typical structure of the giant-cell tumor of bone with numerous characteristic giant cells. Early in December, 1929 the patient complained of pain in the chest; he began to lose weight and his physical condition rapidly deteriorated. A roentgenogram taken at this time showed numerous masses of new growth in both lungs. In March, 1930, he became paralyzed from the waist downwards, and he died on April 3, 1930, or about six years from the appearance of the first symptom.

Fortunately, a post-mortem examination was made in this case. It showed many hard and greatly enlarged glands in the hila of both lungs; both lungs filled with nodular masses of new growth; these were firm in consistency and in many cases formed rounded protuberances from the pleural surface. On cutting into the organs the masses of growth were found to be generally scattered through the pulmonary substance. The tenth rib on the right side showed a diffuse swelling in the mid-axillary line; the bone over this had been reduced to a shell. Both kidneys were enlarged and firm; both showed protuberant masses of growth upon the surface and on section both showed large areas of new growth within the renal substance. There were numerous enlarged glands present in front of the vertebral column extending up as far as the diaphragm; the bodies of the lower dorsal vertebrae were diffusely swollen.

Histological examination. The first sections of the masses of new growth from the lungs failed to reveal the characteristic appearances of the primary growth. Numerous further sections of the lung nodules were examined and among these a proportion was found in which were present the characteristic appearances of the giant-cell tumor. The areas of typical tissue were in every case small and scattered in a field composed of necrotic and fibrosed neoplastic tissue. The active areas of tissue showed the characteristic giant cells.

Microscopical examination of the areas of new growth in the kidneys and also of the mediastinal and preaortic lymph nodes showed exactly the same appearances as found in the lungs. The areas of neoplastic invasion for the most part consisted of necrotic and fibrosing tissue but scattered throughout this occurred small foci of active neoplastic tissue showing the typical osteoclastomatous (giant cell) structure.

Dyke states that while from time to time cases have been recorded in which pulmonary new growths have followed upon the presence of such tumors (giant-cell tumor of bone) Kolodny,[11] in summing up his current state of knowledge of bone sarcomata in 1927, wrote:

Cases of benign giant-cell tumor are cited leading to pulmonary metastasis and death. Two sources of error are revealed by a careful study of such cases. The first is that not in all cases mentioned was the primary tumor a giant-cell tumor, and the second that not in all fatal cases are metastases found to have been present . . . there is no case on record in which histologically a typical giant-cell tumor gave pulmonary metastases of typical giant-cell structure.

Dyke adds that Kolodny appears to have written in ignorance of the cases recorded by Finch and Gleave (1926). The cases of Auge and Roux (1922) and of Turner and Waugh (1933) would be accepted by any but the most critical and many authors have reported cases of supposed pulmonary metastasis of the giant-cell tumor which though not completely proven are "highly suggestive."

The cases mentioned have been referred to in my papers of 1924 and 1927.

In a paper entitled: "Malignant Changes Occurring in Benign Giant-Cell Tumors of Bone," Simmons[12] of Boston, states:

It has been my misfortune to have seen four cases of giant-cell tumor in which death occurred from metastases; two of these cases were in this series, while two others have been registered since 1925. Dr. Codman tells me he has also seen several cases, and I know of others, published, occurring in other clinics.

This observation led Simmons to make a careful study of all the cases of giant-cell tumor that had been registered in the Bone Sarcoma Registry prior to 1925, 116 in number.

TREATMENT

As regards treatment: Simmons states that primary amputation was performed in 13 patients; all living and well five years later. Secondary amputation was performed in 13 patients; 11 were living five years later and 2 had died of metastases. Resection or excision was performed 13 times (excision 4, resection 7, secondary resection 2) including 2 cases in which curettage had failed. The only death occurred in a case in which excision was performed for a giant-cell tumor of the scapula, in which metastases later developed. Curettage as a primary measure was employed in 46 cases; 4 patients died of metastases, and 1 of postoperative hemorrhage. Irradiation was employed as a primary measure in only 8 cases; 6 patients were well five years later, one went on to amputation, and one died six years later of local recurrence and lung metastases. In 13 other cases irradiation was employed in conjunction with curettage; 3 patients went on to amputation, and one died of metastases.

Behring[13] collected 384 cases of sarcoma of the long bones operated upon in the leading hospitals of Sweden during the years 1901 to 1926. The diagnosis was verified histologically in all but 2 cases of a series of 246. Of this group, 27 were classified as giant-cell sarcoma. All were operated upon more or less radically. In all of these cases the diagnosis of giant-cell sarcoma was made histologically by pathologists of large experience in the study of bone tumors. However, no less than 6 of the 27 patients died of metastases. Behring feels that the question of whether giant-cell tumors are always benign must be left open for the present.

The question at once arises: Have we any explanation to offer of why this group of supposedly benign giant-cell tumors undergoes malignant changes causing death by metastases,

while the majority of similar cases having the same clinical, roentgenological and histological characteristics, run a benign course?

The explanation offered by Stone and Ewing[14] in their paper: "An Unusual Alteration in the Natural History of a Giant-cell Tumor of Bone" is, perhaps, the best that has thus far been advanced. While I was not inclined to accept it at the time, after a careful analysis of a group of similar cases (benign giant-cell tumors that have undergone malignant transformation) that have since come under observation, I am convinced that it is worthy of very serious consideration. These authors, in their discussion of Case v state:

> The development of metastases in this case proves no exception to the rule that the benign giant-cell tumor never produces metastases. Here, the metastasizing tumor was not a giant-cell tumor, but a malignant growth that developed out of a giant-cell growth, as the result of various insults, in which curettage and imperfect irradiation probably played the chief part.

The attempt of Stone and Ewing to preserve the rule laid down by Nélaton and Bloodgood, i.e., that giant-cell tumors never metastasize, by stating that the primary tumor in question did not metastasize but that it simply furnished a site for the development of a new malignant tumor that did metastasize, hardly calls for discussion. It is, at most, an academic question without any practical significance to either the surgeon or the patient. I have observed a number of cases in which malignant tumors developed at the site of supposedly light doses of *x*-rays given for benign conditions. If the malignant tumor in this case was caused by the insults of the surgeon and by imperfect irradiation, it is most important for us to determine if possible which of these two factors was chiefly responsible. Of course, it is quite impossible to answer this question without additional information obtained from a study of similar cases. It calls for a group of cases treated by curettage alone as controls, and a group treated by prolonged irradiation associated with curettage.

Fortunately we already have sufficient data at the Memorial Hospital and the Hospital for Ruptured and Crippled to furnish at least a tentative answer to this question. We have a group of 23 cases treated by curettage and prolonged postoperative irradiation, and a group of 31 cases treated by curettage (with or without postoperative toxin treatment) without postoperative irradiation.

A careful analysis of these two groups brings out the striking fact that in no less than 7 of the 23 cases treated by curettage and prolonged postoperative irradiation, the benign giant-cell tumor later underwent malignant transformation, whereas in only a single instance did this change take place in the group of 31 cases treated by curettage without irradiation. To the latter group might be added the Johns Hopkins series of 105 cases treated by curettage without irradiation, in which there was not a single case of malignant transformation.

This analysis would seem to eliminate surgical curettage as the important causative factor in the transformation of a typical benign giant-cell tumor into a malignant metastasizing tumor, and would force us to assign a large measure of the responsibility to irradiation associated with curettage.

I believe these results fully justify the conclusion reached by Stone and Ewing that, "it is unwise to attempt to combine surgical methods with postoperative radiation by means of radium inserted in the tumor cavity." In fact, one might go even further than this, and conclude that it is unwise to combine surgical methods with irradiation of any kind.

In only one of our cases was radium used in the manner condemned by Stone and Ewing, i.e., implanted in the cavity after curettage.

Four of the cases in our series received prolonged irradiation before curettage. In the first (see Case x) a giant-cell tumor of the lower end of the radius was treated by *x*-rays for one and a half years at the French Hospital. The improvement was only temporary. Curettage on three occasions was followed by a prompt recurrence. *Microscopical diagnosis:* benign giant-cell

tumor. A year later I performed an amputation of the arm, and the tumor proved to be malignant osteogenic sarcoma. In the second case (see Case IX) the clinical and roentgenological diagnosis of typical benign giant-cell tumor of the femur had been confirmed by microscopical examination following curettage. Two years later an amputation was performed, and the tumor proved to be a malignant osteogenic sarcoma. For the third and fourth examples see Cases XI and XII.

Is it possible to explain how irradiation associated with curettage may be influential in transforming a benign giant-cell tumor into a malignant metastasizing tumor?

I cannot agree with Geschickter and Copeland that "giant-cell tumors constitute a single entity" or that "the giant-cell tumors arise as an abnormal phase in the reabsorption of tumor bone in response to trauma, while the bone cyst presents a healing phase of this same lesion." While I am in complete accord with those who believe that trauma plays an important part in the development of giant-cell tumors (our series shows a history of antecedent local trauma in 56 per cent, and the Johns Hopkins series in 65 per cent) I would point out that there still remains a considerable group in which there is no history whatever of antecedent local trauma. This being so, how are we to explain the origin of these tumors on the theory advanced by Geschickter and Copeland?

Personally I do not believe that the many different types of bone sarcoma represent separate specific diseases or separate distinct entities. The recent experimental work of Berg[15] in the laboratory of the Hospital for Ruptured and Crippled refutes this. By inoculating the marrow of the tibia of week-old chicks with endothelioma virus obtained from Rhode Island Red chickens, Berg was able to produce a typical osteogenic sarcoma, a plasma-cell myeloma, an endothelial myeloma, a giant-cell tumor, and finally, one epithelial tumor. The diagnosis of these tumors was confirmed by a number of experienced pathologists.

If it is possible to produce in chickens a number of different types of bone sarcoma corresponding to the types found in man, by inoculations of an extrinsic agent or virus, it is not unreasonable to suppose that a similar extrinsic virus or agent might produce the different types of bone sarcoma in man.

There are doubtless many other important causative factors in the etiology of bone sarcoma beside trauma. The type of tumor may depend entirely upon the cells of the host, upon which cell it finally selects for its habitat, and not upon the extrinsic agent. This theory is explained in more detail in my paper on "Some Thoughts on the Problem of Cancer Control."[16]

We have abundant evidence to prove that prolonged irradiation may produce malignant tumors. Most of these occurred in physicians who, in the earlier years, not realizing the dangers to which they were exposed, did not properly protect their hands. While most of these *x*-ray tumors were carcinoma or epithelioma, a number were sarcoma. In one of my own patients, ten years after a severe dermatitis of the thigh following prolonged *x*-ray treatment, there developed three different types of malignant tumor at the site of the dermatitis: a sarcoma, a carcinoma of the soft parts, and a typical epithelioma with pearls in the marrow cavity of the femur. We know from Martland's[17] remarkable study of the skeletal bones of the dial painters in the watch factories of Newark, who died of radium poisoning, that the majority had definite osteogenic sarcoma. Recently Schuch and Wehlinger,[18] by the implantation of radium tubes in the periosteum of rabbits, succeeded in producing typical osteogenic sarcoma after an interval of two years. In one case in which they injected 3 mg. of mesothorium, an osteogenic sarcoma developed in twenty-one months.

These facts would seem to show that the use of prolonged irradiation, especially with gold seeds of radon, in close contact with the bone after curettage for giant-cell tumor, might easily be an important factor in lowering the resistance of the

neighboring normal cells and permit the lighting up into new activity of any latent tumor cells left behind from the curettage; once started, and with this lowered resistance, it is not difficult to understand how this activity might go on increasing until it ended in a malignant tumor.

If our theory is correct then we should in the future refrain from using irradiation in any form, either internally (implantation of gold seeds) or externally (application of pack), in conjunction with the surgical removal of giant-cell tumors by curettage or resection.

Those who hold irradiation to be the method of choice, should insist, as Ewing and Herendeen have long done, that no preliminary surgery (curettage or biopsy) be employed, and that these tumors be treated by irradiation alone. My main objection to this method is that in cases so treated there will always be found an error in diagnosis in from 20 to 25 per cent of the cases, and that by the time this error is discovered it is usually too late to save the life of the patient by amputation. I am strongly opposed to a simple open biopsy in giant-cell tumors of the long bones because of the probability of sinus-formation and the danger of subsequent infection. The objection to primary irradiation without an open biopsy has been partially overcome in recent years by the employment of the aspiration biopsy. This has proved useful in a certain number of cases of giant-cell tumor but only in those cases in which the condition was sufficiently far advanced to cause complete or partial destruction of the bony shell, thus permitting the aspirating needle to penetrate into the tumor.

Personally, I still regard surgical treatment as the method of choice, that is, thorough curettage, chemical cauterization, and closure of the wound without drainage; and my reasons for this are as follows: (1) We avoid error in diagnosis; (2) there is a shorter period of disability, and (3) there is a greater certainty of a permanent cure.

In analyzing the group of cases originally diagnosed as *benign giant-cell tumor* by experienced pathologists, which later

became malignant metastasizing tumors, we have divided our cases according to the method of treatment employed, in the hope that this might throw some light upon the causative factors responsible for the malignant changes.

The first group consists of 8 cases treated by primary curettage in 7 cases, by biopsy in one, followed by prolonged irradiation. In all of these cases the curettage furnished ample material for a careful histological examination of the entire tumor; and in all, a diagnosis of *benign giant-cell tumor* was made. After a trial of irradiation over varying periods up to two and one-half years, evidence of malignancy developed which was confirmed by microscopic examination; and an amputation was performed in 7 cases. The eighth was a large inoperable tumor of the ilium in which the toxins were used after failure of irradiation. The patient remained well for five years and then died of abdominal metastases. Death occurred in 7 of these cases after varying intervals of a few months to five years. Only one of these patients is alive; this is a recent case and only a year has elapsed since the operation.

In 4 cases the patients were treated by irradiation for a long period before curettage or surgery was employed. In 2 cases curettage was done after failure to control the disease by *x*-rays given over two or two and one-half years. The microscopical diagnosis in both of these cases was that of *benign giant-cell tumor*. Both eventually went on to amputation; and microscopical examination of the amputated specimen showed a malignant osteogenic sarcoma. In the third case, a typical osteitis fibrosa cystica, there developed eight and one-half years after an apparent cure by irradiation, a rapidly growing tumor at the same site; an amputation was performed and the patient died a few months later of pulmonary metastasis. The microscopical diagnosis of the amputated specimen was that of malignant osteogenic sarcoma. The fourth patient, with a tumor of the upper end of the humerus, was given irradiation (30,000 mc. hr.) for a condition that was diagnosed clinically and roentgenologically as a malignant osteogenic

sarcoma, but which, on microscopical examination by Ewing and the Bone Sarcoma Registry, was pronounced a *benign giant-cell tumor*. Two and one-half years later the patient's scapula was removed for a large recurrent tumor which was pronounced a malignant osteogenic sarcoma.

We have 3 other cases (Nos. XIII, XIV, XV), 2 of the humerus and one of the femur, in which I performed a primary amputation on the clinical and roentgenological evidence of malignancy and in spite of the microscopical diagnosis of *giant-cell tumor* of the epulis type. In all of these cases the condition proved to be that of malignant osteogenic sarcoma and the patients died of pulmonary metastasis. I believe that these 3 cases represent an error in diagnosis and not a real transformation of a *benign giant-cell tumor* into a malignant osteogenic sarcoma; the tumors, apparently, were malignant from the start. In my opinion, these cases prove that when the clinical and roentgenological evidence is strongly in favor of a diagnosis of malignant bone tumor, more importance should be attached to it than to the histological evidence of *benign giant-cell tumor*.

In 4 other cases, a histological diagnosis of *giant-cell tumor* was made after an operation (curettage and resection); the disease recurred and the patients died of pulmonary metastases. One of these was a sarcoma of the humerus treated by curettage and the insertion of a Lane plate for a pathological fracture. It is possible that the irritation of the Lane plate may have been a causative factor in the development of the local recurrence. The latter proved to be a malignant osteogenic sarcoma. An amputation was performed, but the patient died of pulmonary metastasis. In the second case, a tumor of the lower end of the radius was resected by Dr. Lilienthal who later referred the case to me for a recurrence in the lower end of the ulna. The roentgenogram of both of these tumors showed a typical benign giant-cell tumor, which was the diagnosis made after a microscopical study of the first tumor of the radius. An amputation was performed, but the patient died

of pulmonary metastasis. The sections of the tumor of the ulna showed numerous giant cells.

The treatment of so-called *benign giant-cell tumors* that have already become definitely malignant should be the same as the treatment of primary malignant osteogenic sarcoma of the long bones, namely, early amputation followed by prolonged treatment with the mixed toxins of erysipelas and Bacillus prodigiosus. Unfortunately only one such case in our group received this treatment. This was Case XIII in which I performed an immediate amputation on the clinical diagnosis of malignancy: although, the microscopical diagnosis of *typical benign giant-cell tumor of the epulis type* had been made by a pathologist of considerable experience in bone tumors. This patient died of pulmonary metastasis one year later. This was, undoubtedly, a primary malignant osteogenic sarcoma, and represents an error in diagnosis. No matter what method is employed in these cases, the prognosis is very grave for the reason that the disease is almost always far advanced before its true nature is recognized.

This is hardly the place for a lengthy discussion of the treatment of benign giant-cell tumors in general, but inasmuch as we have shown that the cause of the malignant transformation of these giant-cell tumors is, in all probability, closely associated with errors in the method of treatment employed, it is important to state briefly what we believe to be the most efficient, and, at the same time, the method that is least likely to increase the tendency to malignancy.

At present there is the widest difference of opinion as to the best method of treating benign giant-cell tumors of the long bones. In spite of the teachings of Paget and Nélaton, radical treatment by amputation continued to be the routine method employed not only in Europe but in America up to 1915. I began the conservative treatment of all long-bone sarcomas, including giant-cell tumors, as early as 1897. This consisted of a brief course of injections of the toxins of erysipelas and Bacillus prodigiosus for four or five weeks; if no

improvement was noticed at the end of this time, I proceeded to amputate, following this with further toxins as a prophylactic measure. My first success (1897) occurred in an osteogenic sarcoma of the femur in which the limb was saved, and the patient remains well today, thirty-eight years later. My first success in giant-cell sarcoma (1904) occurred in a giant-cell tumor of the lower end of the tibia, an aggressive type, that had recurred promptly after two curettages; the toxins were combined with local *x*-rays; the limb was saved, and the patient remains well today, thirty-one years later.

My first radical operation (amputation) for a giant-cell tumor was performed in 1895, in a boy aged ten years, with a tumor of the tibia. Since then I have performed an amputation in only 4 cases in which a microscopical diagnosis of giant-cell tumor was made after the amputation. In 3 of these cases I made a clinical diagnosis of malignant sarcoma, and amputated the limb without waiting to do a biopsy; all 3 patients died of metastases, thus justifying the radical treatment. I believe these cases undoubtedly represent errors in histological diagnosis, and not a transformation of a benign giant-cell tumor into an osteogenic sarcoma. In the fourth case, (1915), a rapidly growing tumor of the fibula in which a clinical and roentgenological diagnosis of *malignant tumor* had been made, I did a complete excision of the fibula following this with fulguration of the wound; three days later I was obliged to amputate for gas gangrene. The patient made a good recovery, and is well at the present time, nineteen years later. This case represents not a primary amputation for giant-cell tumor but an emergency secondary amputation.

In 1914, Bloodgood took a strong stand for conservative operation for giant-cell tumors, reviving the early teachings of Nélaton and Paget regarding the benign character of these tumors. He advocated primary curettage with chemical cauterization, and resection for the more advanced cases. At this time practically all the cases at Johns Hopkins were being treated by amputation. A study of the later reports of Johns

Hopkins shows how slow the members of the surgical staff were to adopt the conservative method so strongly and repeatedly urged by Bloodgood: in 1909 there were no cases treated by curettage, but 3 by resection and 4 by amputation; and in 1920, 4 were treated by curettage, 2 by amputation and 3 by resection. Even at the present time not a few cases are being treated by amputation as shown by a recent report of Matz[19] of the U.S. Veterans' Hospital: in 5 cases of giant-cell tumor observed among 45 cases of bone sarcoma occurring in ex-service men, all were treated by amputation; 2 by primary and 3 by secondary amputation.

While conservative methods have largely replaced amputation in this country, conservative surgery (curettage and chemical cauterization) has been gradually superseded by primary irradiation, the method strongly advocated by Ewing, Herendeen and Pfahler of America, and Regaud of France, until today, the majority of giant-cell tumors are, I believe, being treated by irradiation. While there is no doubt that irradiation has been successful in many instances, few radiologists have reported a sufficient number of end-results to permit a comparative study with other groups treated by surgery.

One reason for the marked trend toward irradiation, in my opinion, is the undue emphasis placed upon the bad results of surgery. That they have occurred, cannot be denied; but they have been far less frequent than one would be led to believe from published reports, and, more important, they occurred prior to fifteen years ago when the old surgical technique (curettage followed by packing and drainage) was almost universally employed.

On the other hand, the bad results of irradiation in the treatment of giant-cell tumors are almost never mentioned; and these, in my opinion, have occurred more frequently, and are of a more serious nature than the bad results of surgery, *for example*, severe burns, intractable ulcers remaining unhealed for years and sometimes requiring an amputation, marked

impairment of the function of the joint and late osteomyelitis. One of the Memorial Hospital patients, with a giant-cell tumor of the femur apparently cured by irradiation in 1921, developed a severe ulcer seven and one-half years later; one year later an extensive osteomyelitis developed, which in spite of conservative operation and daily dressings for nearly a year, finally required an amputation. In this case there was complete ankylosis of the knee over the entire time.

At the Memorial Hospital we have had only a single bad result from surgery in fifteen years. This occurred in a case already referred to, in which I did a resection for a very extensive giant-cell tumor of the fibula; unwisely I treated the large open wound by fulguration before closing it. Gas gangrene developed, requiring an amputation three days later; the patient recovered and has been well ever since. In this case the tumor was so extensive and rapid-growing, having all the features of malignancy, that most surgeons would have amputated in the first place. At the Hospital for Ruptured and Crippled, I have had one case of severe infection following curettage for giant-cell tumor of the lower end of the femur, necessitating an amputation. This case also was observed over fifteen years ago. There is still another case in which a biopsy had been performed at another hospital, and the wound had become infected, so that I had to perform an amputation; this patient is well fifteen years later.

I believe the favorable results following the surgical treatment of giant-cell tumors may be attributed to the improved technique, first employed by Dr. Bradley L. Coley at the Memorial Hospital in 1923; this consists of thorough curettage followed by chemical cauterization and closure of the wound without packing or drainage. In not a single instance has the wound failed to heal by primary union.

The Place of Coley's Toxins in the Treatment of Giant-cell Tumors. While I believe that surgery alone, viz. thorough curettage with chemical cauterization (50 per cent chloride of zinc) and closure of the wound without packing or drainage,

will effect a permanent cure of the great majority of cases of giant-cell tumors of the long bones, there will always be a certain number of recurrences. Bloodgood found 30 per cent recurrences in the Johns Hopkins series of 105 cases; most of these were treated by the older method of curettage and packing. I believe that the number of recurrences could be greatly reduced if postoperative treatment with the toxins of erysipelas and Bacillus prodigiosus was given as a routine measure after curretage. It could be started in the hospital and continued at home for two or three months by the local physician. The fact that some of our most advanced and inoperable cases of giant cell tumor have been cured by the toxins alone would seem to prove that such a powerful, inhibitory agent would be useful as a prophylactic against recurrence. If it is not thought necessary to use the toxins in the early cases with little bone involvement, I feel sure they ought to be used in all the advanced cases. A brief analysis of our own series strongly supports this view. Of 31 cases treated by curettage followed by toxins, only 2 have recurred; both of these were of the aggressive type requiring amputation after partial control with toxins and irradiation.

CASE REPORTS

CASE 1. *Benign giant-cell tumor of the femur which later became malignant.*

G. G., male, aged thirty-nine years, while working on May 10, 1928, slipped on a stairway and injured his right knee; pain and swelling developed shortly after. A curettage was performed by Dr. I. Zadek, who packed the wound with gauze and applied a posterior splint.

Roentgenograms and microscopic sections were examined by Dr. Ewing on February 15, 1929, who reported as follows: "The radiographs show that on May 10, 1928, the day of the injury, this man had a well-established giant-cell tumor of the femur, which could not possibly have been produced by the injury occurring on that date. The subsequent radiographs show a steady increase in the dimensions of this tumor, in spite of radiation. While nearly all of these tumors may be cured by radiation, if not operated upon, yet many of them prove resistant after curettage. The structure of this tumor is entirely benign and incapable of producing

pulmonary metastases, but the recurrence may be more malignant and radioresistant. From the study of the last *x*-ray taken January 28, 1929, I think it ought to be possible to control this growth by proper radiation

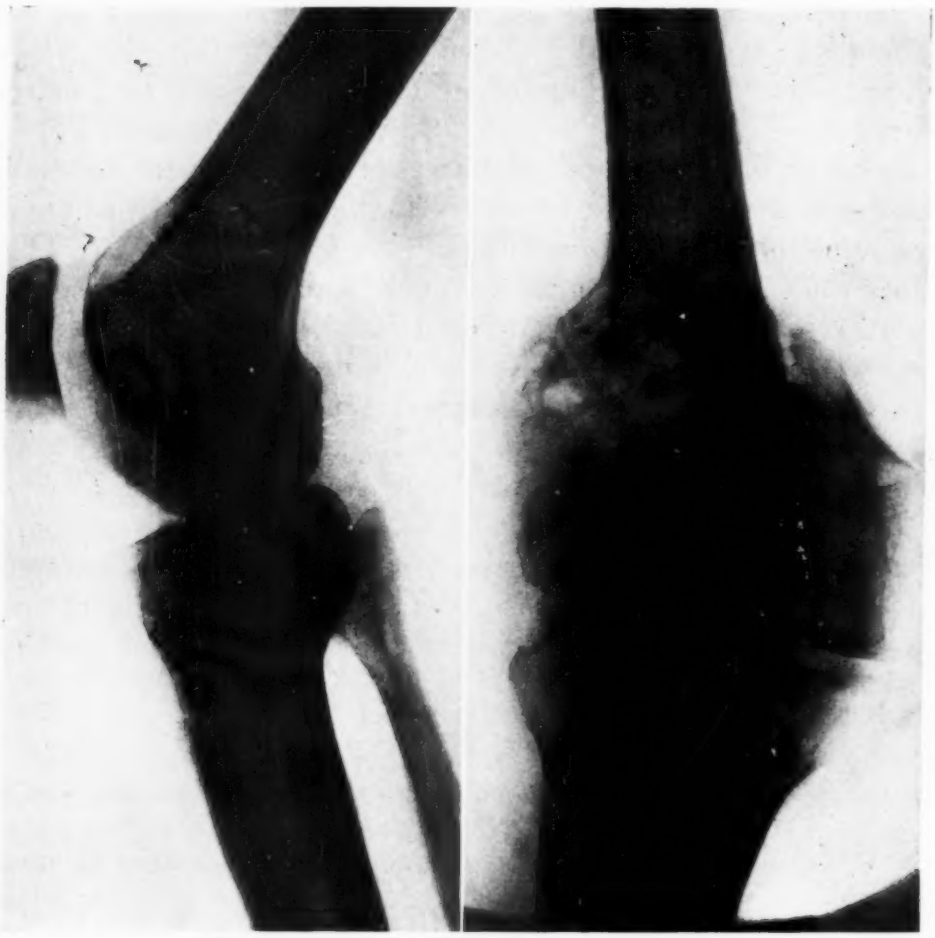

FIG. 1. FIG. 2.

FIG. 1. Case 1. Benign giant-cell tumor of femur. May, 1928.
FIG. 2. Same case, three years later (February, 1931).

and I recommend that this treatment be persisted in."

X-ray treatment was begun on March 7, 1929 by Dr. Herendeen, and continued, with intervals, until May, 1931. In the fall of 1930, a pathologic fracture developed. There was more or less ankylosis and poor alignment, and in December, 1930, the patient was admitted to the Hospital for Ruptured and Crippled where an attempt was made to straighten his leg sufficiently to permit him to get about on it. At this time there was no

definite evidence of active disease either clinically or by roentgen-ray examination. Under general anesthesia the fragments were arranged in better position, and the leg was put in a plaster cast. When the latter was removed, inspection showed no great improvement in the alignment. I urged an immediate amputation as I believed that even if the disease were cured, the patient never would have a useful limb. On April 20, 1931 he was readmitted to the Memorial Hospital, and while walking about the wards, he fell, sustaining a compound fracture at the site of the old lesion. An immediate amputation was performed by Dr. F. E. Adair.

The most remarkable feature of this case is, that a study of the lower end of the femur after amputation showed, instead of a healed or an entirely controlled benign giant-cell tumor as supposed, a malignant osteogenci sarcoma.

Patbological report by Dr. F. Stewart, June 2, 1931: "Specimen leg amputated at mid thigh region. Evidence of old pathological fracture near the epiphysis of femur with subsequent necrosis and ulceration, anterior and lateral portion of leg measuring about 10 cm. in diameter. Soft part tissue was removed from knee region down to mid portion of lower leg exposing the tibia and fibula and considerable necrotic tissue in region of knee joint. Lateral portion of the tibia and in the region between the tibia and fibula is considerable rather firm soft-part tumor tissue (gross diagnosis) from which a section was taken. Tibia and fibula sawed through about the mid portion and placed in formalin. *Microscopical:* Very cellular osteolytic osteogenic sarcoma."

A follow-up note by Dr. Adair on September 12, 1932 states that there is a small mass in the right groin. An aspiration biopsy was done on the following day, and the specimen pronounced a malignant tumor. Further *x*-ray treatment was given from September 22 to February 7, 1933. Examination on November 18 showed a mass lying directly over Poupart's ligament and extending down over the femoral opening. The patient complained of intense pain and on March 16, 1933 he was operated upon at the Neurological Institute (thoracic hemilaminectomy with cordotomy). He was readmitted to the Memorial Hospital where a roentgenogram taken on May 3 showed evidence of pulmonary metastasis. Death occurred on May 11, 1933 more than four years after the tumor was first noticed.

This case I believe is one of the most convincing presented in proof that a primary benign giant-cell tumor (so pronounced by a number of experienced pathologists) may later become a malignant tumor and cause death by metastasis.

It is interesting to note that in Dr. Ewing's original report on the primary tumor he stated: "While nearly all of these tumors may be cured by radiation, if not operated upon, yet many of them prove resistant after curettage. The structure of this tumor is entirely benign and incapable of producing pulmonary metastases, but the recurrence may be more malignant and radioresistant."

It is possible in this case that prolonged irradiation after curettage, together with forcible extension of the leg in an effort to get better alignment after the pathological fracture, may have been an important factor in bringing about this malignant change.

CASE II.

S. B., male, aged twenty-three years, was admitted to the New York Hospital in March, 1918, where he was treated by curettage of the head of the tibia for an osteomyelitis. *Microscopical examination* showed one portion to consist of spindle-cell sarcoma, and another to contain numerous giant cells of the epulis type in a spindle-cell stroma. The patient was then transferred to the Memorial Hospital, April 23, 1918, where he received one application of the radium pack (14,940 mc. hr. at 6 cm. distance). He was lost track of for several years. In December, 1923, a roentgenogram taken at the Memorial Hospital showed an old osteomyelitis in the head of the left tibia; no evidence of active process.

On September 27, 1932, he struck his knee against an ash can; intense pain followed. He was admitted to the Presbyterian Hospital where, on October 11, 1932, a partial ostectomy of the tibia was done, the old cavity being curetted out and new bone fragments being used to fill it. In curetting, the surgeon found the fragments of bone used fourteen years ago together with some wax. *Microscopical diagnosis:* osteitis fibrosis cystica and fibrosarcoma of tibia.

Following the operation there was a marked reaction, apparently due to an invasion of the surrounding tissues by the neoplasm. A biopsy was done. Microscopical diagnosis: "fibrosarcoma of tibia (recurrent). These sections show a considerably less differentiated growth than the original in the tibia, and represent a very marked grade of malignancy." As a result of this, an amputation was performed on November 18. *Microscopical diagnosis:* fibrosarcoma of tibia. A roentgenogram taken on November 16, 1932, failed to show any evidence of bone involvement other than the primary lesion. The patient was discharged on December 7, 1932 in good

condition except for the loss of his lower extremity. Several months later he was admitted to the Morrisania Hospital where a gland was removed, and the following *microscopical* report received: "Gross description: gland enlarged; feels firm. There are small hemorrhagic areas present throughout gland. Microscopic description: Section shows spindle-cell sarcoma." A roentgenogram taken at the same time showed: "Mottled infiltration throughout both lungs; a few lesions appear discrete and resemble metastatic processes." The patient died on January 14, 1933.

CASE III. *Giant cell sarcoma of ilium. Curettage and prolonged irradiation. Recurrence; condition inoperable. Treated with toxins for four months. Curettage. Remained well for five years, and then died of metastases.*

Mrs. F., aged thirty years, fell on the ice in 1908 receiving a severe blow on the buttock and ilium. Two months later a tumor developed at the same site. An operation was performed by Dr. Andrew J. McCosh at the Presbyterian Hospital, and a *microscopical diagnosis* of giant-cell sarcoma was made. X-ray treatment was given shortly after the operation. The tumor recurred and became inoperable. Radium treatment was begun in June, 1909 by Dr. R. Abbe of New York, and continued during 1910, in large doses, by Dr. Wickham of Paris. The patient was referred to me by Dr. Frank Hartley in May, 1911, at which time she was markedly anemic and considerably emaciated, suffering from intense pain. Physical examination showed a large inoperable tumor of the ilium. Under four months' toxin treatment, the tumor became very much smaller and broken down, making it possible to do a curettage.

Microscopical report by Dr. Ewing: "The tumor, diagnosed as giant-cell sarcoma, proves in my sections of the material to be, as supposed, a giant-cell sarcoma. It is composed chiefly of small spindle cells in which lie many giant cells of the epulis type. There are numerous areas of hemorrhage, and the giant cells are most numerous in these areas. The structure is that of a tumor of moderate malignancy. Its position may render it more serious than if it were in a superficial position, but histologically, it is not to be classed with the more malignant or periosteal growths."

The patient, apparently, made a complete recovery and remained well for five years, when the disease returned, and she died in the following year of symptoms of probable intra-abdominal and pelvic metastases.

CASE IV. *Benign giant-cell tumor of tibia treated by repeated curettage and prolonged irradiation. Disease not controlled. Amputation. Local recurrence. Death from metastases.*

J. S., female, aged forty-four years, was operated upon at the New York Hospital by Dr. Pool, for a central tumor of the upper end of the tibia (curettage and implantation of fat) in the latter part of October, 1920. *Microscopical diagnosis* by Dr. Elser: giant-cell sarcoma. He added: "The

structure of this tumor suggests that it is not the usual type of central giant-cell sarcoma of bone. The embryonal character of the unicellular elements, the abundant of mitotic figures, some of which are atypical, the excellence of nutritional condition (practical absence of necrosis and hemorrhages) differentiates this growth from the usual central giant-cell sarcomas. The growth bears all the evidence of malignancy and the early recurrence is not surprising. The case illustrates the fallacy of regarding all of these growths benign and curable by simple curettage. Certain tumors of this class are not only locally malignant, but give rise, though rarely, to generalized metastases."

The tumor recurred locally and a second operation was performed by Dr. Pool in November. This was examined microscopically and again pronounced a giant-cell tumor. In this diagnosis Dr. Ewing concurred. The patient was then transferred to the Memorial Hospital where irradiation was given over a period of eight months. In spite of this the disease continued to progress, and an amputation was performed by Dr. Auchincloss at the Presbyterian Hospital on October 26, 1921. *Microscopical diagnosis:* giant-cell tumor of the epulis type. The disease recurred in the stump, and a few months later it extended into the pelvis and spine, causing death on November 5, 1922. (For full history see my previous report.[19a])

CASE V. *Benign giant-cell tumor of tibia. Curettage (by Dr. Martin at St. Luke's Hospital) and implantation of unfiltered radon seeds in cavity (47 mc. for 48 hours). Slow healing without infection. Recurrence eleven months later; hemorrhage and infection. Amputation in June, 1920. Evidence of pulmonary metastases in January, 1921; death a few weeks later.*

J. N., male, aged nineteen years. This case has already been fully reported in my paper[19a] and by Stone and Ewing[13] and by Geschickter and Copeland,[7] and will receive only a brief mention here. Suffice it to say, the microscopical diagnosis of the primary tumor by Ewing, Bloodgood, Stewart of Leeds and the Committee of the Bone Sarcoma Registry, was benign giant-cell tumor. Following the amputation, Dr. Ewing reported: "Section shows a large spindle-cell sarcoma with much edema and widely dilated blood vessels. It resembles malignant osteogenic sarcoma."

As this patient was on my service at the Memorial Hospital, I am familiar with the clinical and pathological findings from the beginning. I am still of the opinion that this was, as Ewing and Stone believed, a true example of a primary benign giant-cell tumor which later became a malignant osteogenic tumor. The explanation of this change offered by Ewing and Stone is discussed elsewhere.

CASE VI. *Giant-cell tumor of the femur (diagnosis based on clinical and roentgenological evidence supplemented by microscopic sections from biopsy). Not controlled by prolonged irradiation. Metastasis to lungs. Death.*

F. S., female, adult. In this case an exploratory operation was performed at an outside hospital, and the diagnosis of giant-cell sarcoma made from the microscopic sections. The patient received irradiation (3 applications of radium needles, and 3 exposures to *x*-rays) at another hospital before entering the Memorial Hospital on March 10, 1923 for further irradiation. *Roentgenological diagnosis*, Dr. Herendeen: Giant-cell sarcoma of distal end of femur. The tumor rapidly increased in size and was accompanied by intense pain. An amputation was performed, and on *microscopical examination* a diagnosis of highly malignant spindle-cell sarcoma was made. Pulmonary metastases developed and the patient died a short while later.

In this case it is impossible to state definitely whether the tumor was originally a malignant osteogenic sarcoma, erroneously diagnosed as a giant-cell tumor, or whether it was primarily a benign giant-cell tumor which was subsequently transformed into a malignant osteogenic sarcoma.

CASE VII.

E. M., female, aged eighteen years. No history of trauma. In January, 1928, the patient first complained of pain below the right knee, impairment of motion at the knee, and a swelling about one inch below the joint. In the middle of February, a local operation was performed at an outside hospital. She remained well for three weeks and then had a secondary hemorrhage. The wound was packed and she was given a transfusion. On March 20, 1928 she was admitted to the Memorial Hospital, on the service of Dr. Adair. Physical examination at this time showed the right leg flexed at the knee to an angle of about 85 degrees. A provisional diagnosis of *giant-cell sarcoma* was made.

Radiographic report by Dr. Herendeen, March 22, 1928: "The upper end of the right tibia is involved in a destructive process which presents in the films the features of a malignant bone tumor, possibly medullary in origin. X-ray diagnosis: medullary and cortical osteogenic sarcoma. It is of course conceivable that infection of a giant cell tumor which has recurred after a curettage could present similar features. It is believed, however, that the process now is malignant."

On consultation with Doctors Ewing, Herendeen and Craver, it was decided to give irradiation by high voltage *x*-rays and radium pack. This was done after removal of the packing (the wound had been packed daily with gauze). There was a large cavity in the head of the tibia. The tumor

tissue soon filled this cavity and fungated out in the surface of the skin. The tumor seemed not to be controlled by irradiation although the doses were heavy enough to markedly excoriate the skin. Dakinzation to control the infection was unsuccessful. Daily applications of chloride of zinc paste were used in an attempt to restrain the growth, but this was only temporarily effective. On May 26, 1928, the leg was amputated at the mid-thigh. A roentgengram taken on May 18, had shown definite evidence of metastases in the lungs. Death occurred on August 18, 1928, or less than three months after the amputation.

The following pathological reports were made at the Memorial Hospital:

"*March* 28, 1928, No. B-4379. *Gross:* Specimen from fungating infected recurrent giant cell tumor of tibia. *Microscopical:* The general structure is that of the typical giant cell tumor of epulis type. The giant cells are very large and very numerous, and they line capillaries or lie in the stroma. The stroma cells are not numerous but they are rather large and hyperchromatic. Intercellular material is scanty, but in places it approaches the hyaline or even perhaps osteoid type. There is no definite bone production. *The diagnosis* is benign giant-cell tumor of intermediate type such as recurs after curettage.

"*April* 25, 1928, B-4599. *Gross:* Specimen from knee; is 3 by 1.5 cm. and less than 1 mm. in thickness. In places it is hemorrhagic, others composed of dense white tissue suggest necrosis by paste. *Microscopical:* Remnants of giant-cell tumor in areas of hemorrhage and necrosis.

"*May* 26, 1928, B-4815. *Gross:* Specimen consists of right leg the upper third of which in its anterior portion is occupied by an ulcerated necrotic tumor mass. The necrosis is apparently complete in all of the superficial aspects. The ulceration extends into the tibia completely destroying it in the upper 10 cm. with the exception of cortex on the medial side. The tissue here is reddish brown and may be the product of hemorrhage and infection. The patella and fibula seem free from disease. Viable tumor tissue has perforated the cavity of the knee-joint and a small nodule is present posteriorly in the floor of the popliteal space. Dissection of the large blood vessels discloses no tumor tissue. Malignant primary bone tumor. *Microscopical:* Malignant giant-cell tumor. Many giant cells, mostly atypical, some with mitotic figures, many large stroma, spindle cells, hyperchromatic."

A report of this case was not sent to the Bone Sarcoma Registry until 1933, so that the committee had the advantage of knowing the end-result (death from metastases) when they submitted their opinions. Nevertheless, the latter are of a good

deal of interest as bearing on the present problem, and I quote them as follows:

Date	*Diagnosis*	*Rendered by*
1–20–33	Registered as an osteogenic sarcoma. Whether it was ever purely a giant-cell tumor I have some doubt. Am more and more convinced that when there is doubt as to the benign character of a bone lesion from radiologic, clinical or histologic viewpoint, it should be treated as a malignant one—and let the disputes follow.	B. C. Crowell
3–16–34	Osteogenic sarcoma. Some of the giant cells in this section are of the foreign body type but others are of the tumor type.	C. C. Simmons
3–13–34	The three slides submitted show many mitoses and cells resembling but not exactly like foreign body giant cells. The *x*-rays from the first are not at all like those of a giant-cell tumor for the destruction does not proceed to the articular cartilage. There was a mistake in diagnosis rather than a case of giant-cell tumor becoming malignant. Osteogenic from the first.	E. A. Codman
3–21–34	Just like these other cases in lower femur and upper tibia; I am beginning to suspect every case at the upper end of the tibia when clinically or pathologically aggressive looking whether we have to call it giant-cell tumor or not. This is like the H. C. case (see Case IX in present series).	F. W. Stewart
4–16–34	Osteogenic sarcoma. Whether this occurred on base of a benign giant cell or not it is difficult to say. The original *x*-rays are not so sharply circumscribed as we would expect in a benign giant-cell tumor.	J. J. Morton
5– 5–34	Both tumor and foreign body giant cells are present in abundance. The stroma cells are the same as those seen in benign giant cell tumor except there are many mitotic figures. Osteogenic sarcoma (probably malignant change in benign giant cell tumor).	F. W. Hartman

CASE VIII. *Benign giant-cell tumor of the acetabulum treated by curettage, irradiation and toxins. Pulmonary metastases in one year; death.*

A. S., male, aged thirty-four years, was admitted to the Memorial Hospital, service of Dr. Bradley L. Coley, on February 27, 1931, with the following history: A tonsillectomy had been performed in September, 1930. At about the same time the patient developed a limp with marked pain in the left hip. Roentgenograms taken on December 9, 1930 showed a rarefaction of the ilium in the region of the acetabulum. The patient entered the Crown Heights Hospital on December 11, 1930, where an incision was made in the left inguinal region for what was believed to be an abscess. A tumor was found, and some tissue removed. *Microscopical diagnosis*, Crown Heights Hospital: giant-cell sarcoma of ilium. Six deep *x*-ray treatments were given at the Brooklyn Cancer Hospital between December 27, 1930 and February 7, 1931, after which the patient came to the Memorial Hospital. His general condition on admission was only fair; he had lost considerable weight. The left inguinal region was occupied by a swelling which extended into the left lower quadrant, and appeared to arise deeply from the left side of the pelvis. An aspiration biopsy was performed on March 6, and the following *microscopical report* received from Dr. F. W. Stewart: "Smears. Necrotic tissue. Leucocytes. Many giant cells of type found in benign giant-cell tumors."

At the same time, Dr. Stewart examined the sections from the Crown Heights Hospital, and reported as follows: "Unusual tumor. Am inclined to regard it essentially a giant-cell tumor. The rapid increase in size may be the result of softening at the acetabulum and a breaking through of the femoral head with consequent disturbance of the tumor. However, the structure is not wholly typical. There is cartilage, and myxomatous and some new osteoid bone formation (reactive?). Would suggest treating the patient as for a giant-cell tumor keeping in mind malignant possibilities."

Dr. Ewing examined the same sections and reported: "Variant of a giant-cell tumor."

Roentgen-ray examination, February 27, 1931: "No evidence of metastasis seen in films of the lungs. Stereoscopic views of the pelvis reveal evidence of a destructive process involving the left ilium, pubis, and ischium for a considerable distance about the acetabulum. Through pressure, the femoral head has been displaced forward. This tumor appears to be medullary in origin. No other areas of destruction are seen in these films." (No diagnosis made.)

From the time of his admission until May 6, 1931, the patient was treated with high-voltage *x*-rays, and in addition he received thirteen

injections of Coley's toxins. He continued to grow worse, developed metastases, and died in September, 1931.

In this case the diagnosis of Bone Sarcoma Registry (case

FIG. 3. FIG. 4.

FIG. 3. Case IX. Giant-cell tumor of lower end of femur. 1929.
FIG. 4. Same case, two and one-half years after irradiation.

No. 1300) was: giant-cell tumor, malignant variant. (The report of the committee was made after the end-result was known.

CASE IX. *Benign giant-cell tumor of the femur (clinical, roentgenological and microscopical diagnosis). Treated with x-rays for two and one-half years. Pathological fracture. Curettage. Recurrence. Amputation. Death from pulmonary metastases three months later.*

H. C., male, aged twenty-seven years; family history negative; no history of antecedent local trauma. In October, 1928 the patient first felt pain in his left knee. This was regarded as rheumatic and was so treated.

FIG. 5. Section of recurrent osteogenic sarcoma two months after curettage for benign giant-cell tumor. January 14, 1932.

The joint was somewhat swollen and inflamed, and motion was very painful. The inflammation and swelling subsided but the pain recurred at frequent intervals. When the pain was severe he was unable to flex the knee fully, but had little difficulty in walking. The pain seemed to be almost entirely in the popliteal region. In May, 1929 the patient consulted another

physician who had roentgenograms made. These showed a bone cyst or growth, and an operation was advised.

On June 1, 1929 the patient was admitted to the Out-patient Depart-

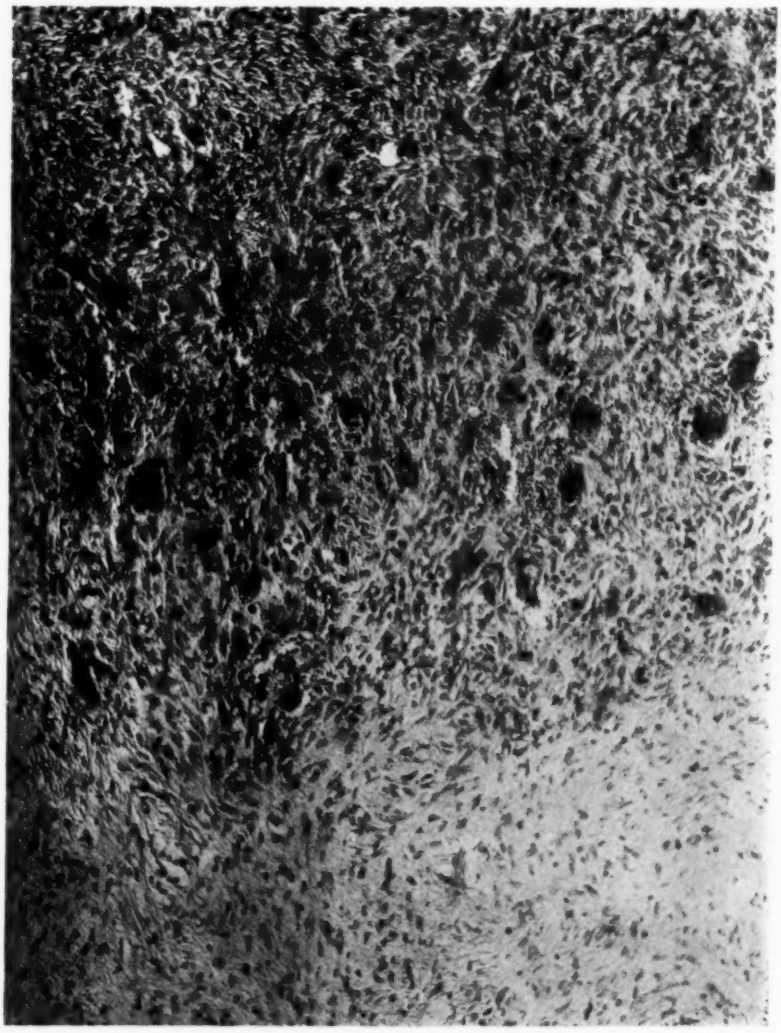

FIG. 6A. Low power photomicrograph. Case IX.

ment of the Memorial Hospital. Examination showed a well-developed, well-built young man of robust appearance. The right knee and left knee were compared and no abnormality in the outline or structure could be determined. There was limitation of motion of the left knee, but no tenderness by deep palpation. The roentgenograms which he presented show a multilocular cystic growth occupying the medulla of the lower extremity

of the femur. Provisional diagnosis: giant-cell tumor of the lower end of the femur.

Beginning June 4, 1929, the patient received roentgen therapy under

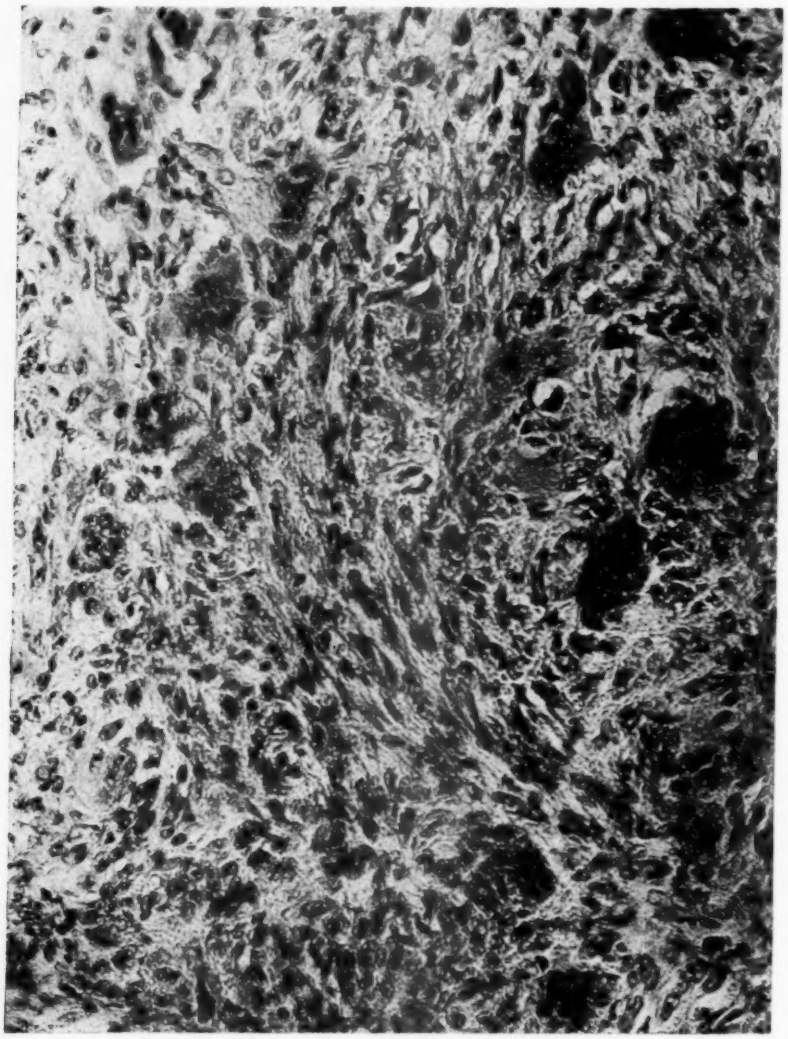

FIG. 6B. High power photomicrograph. Case IX.

the direction of Dr. Herendeen. Roentgenograms taken by the latter over a period of two and one-half years showed a gradual extension of the tumor with increasing destruction of bone in all directions. The tumor at all times, however, presented the appearance of a typical benign giant-cell tumors. On January 18, 1930 a pathological fracture occurred at the lower end of the femur. On February 28, 1930 the leg was put up in a Balkan

frame by Dr. Bradley L. Coley. Examination at this time showed marked improvement; while there was no noticeable regression of the local swelling, the latter was much less tender to touch and movement. A later note on

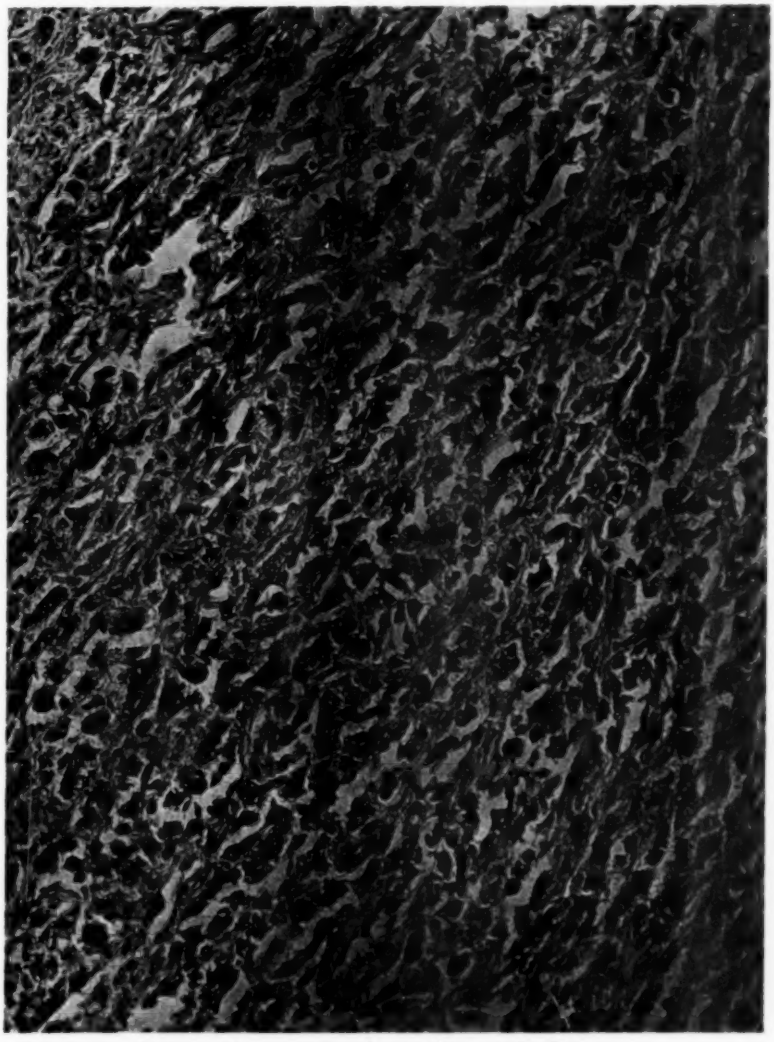

FIG. 7A. Low power photomicrograph. Case IX.

April 30, 1930 stated that the patient's condition was very satisfactory; he was walking with a caliper splint, without pain.

In October, 1930 the patient was referred to the Hospital for Ruptured and Crippled for physiotherapy. He was examined by Dr. Bradley L. Coley on April 8, 1931, at which time there was evidence of slight improvement,

especially as regards increased range of motion. The patient received permission to do a little light work in his brother's shop. Examination on August 19, 1931 showed very little change. There was flexion of about

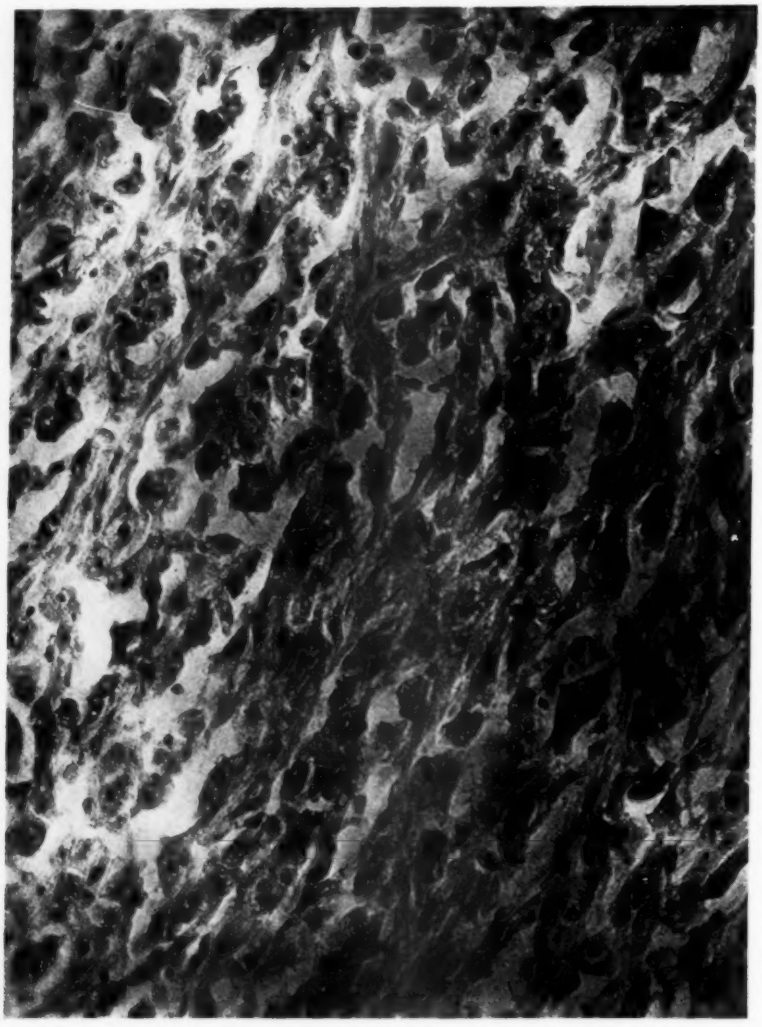

FIG. 7B. High power photomicrograph. Case IX.

20 degrees.

Later note, October 7, 1931, by Dr. William B. Coley: Examination shows no improvement; poor alignment; ankylosis. Believing that the leg will never permit sufficient weight-bearing to be of value, I again strongly advise an amputation.

The patient then consulted Dr. Fred H. Albee, and the following notes are kindly supplied by him*:

H. C. was admitted to the New York Post-Graduate Hospital on October 21, 1931, and was operated upon the next day as follows:

"Through an anterior external incision, with a large curet the tumor material was removed. The bone was egg-shell. The lower end of the femur then collapsed by lateral pressure, as well as shortening of the limb, and a fat fascia graft was put in from the lower thigh region. The skin was closed in the usual manner and a spica applied to the leg."

Pathological report by Dr. Joseph S. Grewal, October 26, 1931: "*Gross:* Specimen consists of a large amount of curettings weighing about 250 gm. They are made up of large bits of soft friable pale gray tissue in places appearing necrotic, mixed with a very small amount of blood. On sectioning some of the larger pieces are made up of rather homogeneous shining tissue showing areas of hemorrhage and some yellowish areas of necrosis.

"*Microscopic:* Sections of the tissue reveal a very cellular structure consisting of somewhat spindle-shaped large cells containing numerous multinucleated giant cells. The spindle-shaped cells contain a centrally located, somewhat vesicular oval-shaped nucleus showing marked changes in appearance and staining qualities. The cytoplasm of these cells also takes on a slightly blue stain. At some places, however, this very cellular tissue gradually changes over into a more fibrous tissue. Distributed through some of the pieces there are small islands of bone formation. Irregular areas of necrosis are also present. The cellular portion with spindle-shaped cells shows fairly numerous mitotic division figures.

"*Diagnosis:* Giant-cell sarcoma.

"*Note:* Because of the evidence of very active growth, a local recurrence of this tumor should be expected. This should be carefully watched for. Eventual amputation may be required."

In spite of the prolonged irradiation which the patient had received before entering the N. Y. Post-Graduate Hospital, the wound healed by primary union, and he was discharged in a satisfactory condition.

About a month later, the patient began to run a fever of from 102° to 104°. He was re-admitted to the N. Y. Post-Graduate Hospital in early January, 1932, and numerous clinical and laboratory examinations were made in an effort to learn the cause of the abnormal temperature. During this period, the lower end of the femur at the site of the previous operation began to show evidence of increase in size, and it was decided that the temperature was due to a rapid recurrence of the disease. It soon became

* I am also greatly indebted to Dr. Albee for the specimen and microscopic sections from which the illustrations are taken.

evident that amputation was unavoidable, and this was performed on January 14, 1932.

Pathological report by Dr. Joseph S. Grewal, January 18, 1932: "*Gross:* Specimen is left leg amputated at the junction of the upper and middle thirds of the femur. The entire tissue around the knee joint and around the distal third of the femur is greatly swollen and fluctuant. On the anterior surface of the knee joint there is a deep scar which is rather irregular in outline and measures about 150 mm. in length. This portion of the tissue is about 220 mm. in diameter and on making longitudinal incision throughout the bone and the soft tissue a large amount of rather turbid yellowish fluid is obtained filling up the entire space of the knee joint including the distal end of the tibia which is broken down and necrotic. Lying underneath the skin and infiltrating underneath the muscle there is a large amount of rather soft pale yellow friable tissue. The distal end of the femur is included in the tumor tissue. It has been completely destroyed with the exception of a very small portion of the articular end which appears as only a cartilaginous shell, measuring about 30 × 40 mm. in diameter and about 3 mm. in thickness. The entire bony tissue of the femur is about normal in thickness but the cortical portion is somewhat thinned out and the marrow cavity is filled with a large amount of fat showing a definite atrophic change. A portion of the joint surface can still be recognized although the upper end of the tibia is covered by tumor tissue which is firmly adherent to the cartilaginous surface. At one place there is a small break in the articular surface, but no definite invasion of the bony tissue can be recognized.

"*Microscopic:* Sections of the tissue taken from the soft gray friable portion of the tumor reveal a rather cellular structure consisting of cells which vary considerably in shape and size. These cells contain large vesicular nuclei with a well-defined nucleolus. Some of the cells have a small amount of pale pink-staining cytoplasm in which at places slight fibrillar substance can be recognized. In other cells the cytoplasm is rather deficient. Mitotic division figures are numerous. A large number of atypical mitoses are also seen. On the whole the cells contain only a single nucleus but occasionally a very large cell with more than one nucleus can be recognized. Larger areas of necrosis are also seen. The entire tissue is greatly edematous. The cells do not show any definite arrangement. In one of the sections the cells form a large amount of fibrillar material. These cells are somewhat spindle-shaped and form interlacing bundles.

"*Diagnosis:* Rapidly growing giant-cell sarcoma of the femur with complete destruction of the lower third of the femur and extension into the knee joint. Edema of the entire leg."

In the early part of February, 1932, a recurrence was noticed in the stump of the leg; this grew very rapidly and was operated upon on February 16, 1932. At the same time a roentgenogram of the chest showed definite evidence of pulmonary metastasis.

Pathological report by Dr. L. H. Meeker: "*Gross:* First specimen measures 11 × 75 × 50 mm. One surface is covered by thickened skin. Across this surface there is a linear wound healed at one end and open for the rest of its extent. Outside the wound area the skin shows pitting due to suture material. The open lips of the wound lead into a cavity with hemorrhagic granulation tissue walls. Beneath the granulation tissue the walls show a peculiar semi-translucent, semi-opaque type of tissue which is fairly well demarkated from the surrounding fat. These areas extend to within a few millimeters of the fascia that covers the lower surface of the specimen. Near the margin of the specimen the skin shows a second linear scar which is open. This also leads into the new-growth tissue just described and is clearly demarkated from the fat in part. At the base near the surface of excision the tissue is firmer and gray, suggesting infiltration by new growth. The consistency of the various structures beneath the skin varies from thick friable tissue in the sinus wall to soft gray areas well demarkated from the fat and to firmer deeper gray areas at the base of the specimen. Second piece measures 65 × 30 × 30 mm. It is similar to the first piece and contains a cavity with necrotic walls. Third piece measures 32 × 20 × 12 mm. It is chiefly fat.

"*Microscopic:* Sections have a covering of stratified squamous epithelium that shows irregular downward prolongations but is everywhere sharply demarkated from the underlying stroma. The supporting stroma shows extensive fibrous tissue proliferation and the hairs and sweat glands are deeply embedded. The walls of the sinuses described in the gross are lined by necrotic tissue which is formed in part by granulation tissue forming large dense collections in some instances and in other instances spreading irregularly beyond the main masses. The neoplastic cells are of various sizes and shapes, polyhedral and spindle forms predominating. The nuclei of these cells also vary in size, shape and staining properties. Most of the nuclei are fairly large and pale with distinct nucleoli. Occasional neoplastic cells show several nuclei and are of giant size. The cytoplasm stains pale pink for the most part but many of the cells take a bluish color. Mitotic figures are numerous and degenerating necrotic cells are especially evident. There is scarcely any reticulum between the neoplastic cells but blood spaces with poorly demarkated walls are scattered throughout. Surrounding the neoplastic areas for the most part there is extensive fibrous tissue proliferation. The fibrous tissue is very edematous, in part very recently formed and in part dense and hyaline. Fibrous tissue strands

extend throughout the fatty tissue and into the underlying muscle. There are large blood vessels in some of the fibrous tissue strands. These show markedly thickened walls in some instances. The fibrous tissue and fat are diffusely infiltrated by lymphocytes, plasma cells and polynuclear leucocytes. In the deeper part the fibrous tissue and muscle are undergoing necrosis.

"*Diagnosis:* Recurrent giant-cell sarcoma.

"*Note:* The malignant characteristics of the neoplasm are not so marked as in the previous specimen."

Sections of the tumor as well as one-half of the amputated leg were given to the Memorial Hospital. These were examined by Dr. F. W. Stewart, Associate Pathologist of the Memorial Hospital, whose report reads as follows:

"*Material obtained at first operation:* This is an aggressive giant-cell tumor. Its structure clearly shows that it belongs in the giant-cell tumor class and yet it possesses certain features which stamp it as locally very aggressive and not entirely benign. These features may be summarized as follows: (1) Many areas are very cellular. The cells are small polyhedral or spindle cells and in these areas the epulis giant cells are very scanty or absent. In other words it emphasizes Nélaton's rule that the fewer the giant cells and the more cellular the "stroma" the more aggressive the tumor. (2) In some areas the stroma cells are long and spindly and tend to form interlacing whorls. (3) There is some scanty deposit of atypical osteoid material. (4) The tumor is telangiectatic. We might therefore conclude that although this lesion is essentially a giant-cell tumor, even at the first biopsy it was already aggressive and showing some malignant features. The presence of these characters in no way lessens the significance of the profound structural alterations found in the amputated specimen.

"*Amputated leg:* Mid-thigh amputation. There is a pathological fracture of the lower end of the femur with complete destruction of the shaft and articular surface. The bone is markedly displaced and over-riding. The joint cavity is invaded by an opaque homogeneous grayish white solid tumor which is secondarily attached to the articular surface of the tibia with some erosion of the latter. The tumor extends posteriorly into the soft parts and forms in all a mass 13 cm. in diameter and 10 cm. in length. Only half of the specimen available for examination.

"*Gross diagnosis:* Osteogenic sarcoma.

"*Microscopically* the sections have lost all the features of benign giant-cell tumor aggressive or otherwise. Nevertheless with the previous sections at hand it is possible to trace certain details of the malignant change. The tumor is now clearly an osteolytic malignant bone tumor. It is made up of atypical hyperchromatic spindle and polyhedral cells lying in a fibrous

stroma. The latter is diffuse and not abundant. Interspersed with the spindle and polyhedral cells are many giant cells. It seems possible to trace transitions from giant cells preserving some characters of the epulis type to typical malignant tumor giant cells with large atypical hyperchromatic nuclei showing marked variations in size and shape within the individual cells, and, in addition, multiple pathological mitotic figures. It would therefore appear that not only the spindle and polyhedral stroma cells of the first tumor but also the epulis giant cells had undergone a malignant transformation."

CASE X. *Benign giant-cell tumor of radius, recurrent after irradiation and repeated* (6) *curettage. Amputation. Microscopical diagnosis: osteogenic sarcoma.*

D. K., female, aged twenty-eight years, was first seen by Dr. Lewis G. Cole on January, 1931, who made a diagnosis of giant-cell tumor of the right wrist. From January 22 to February 24, she was given sufficient radiation to form an erythematous collar about the wrist. She returned to Doctor Cole regularly for observation. In November, 1931, there was apparently a roentgenological and physical reactivation or exacerbation of the pathological process, and at this time another erythema collar was formed about the wrist. She failed to respond, and an operation (curettage and carbolic acid) was performed. No irradiation was used at this time as the erythema collar about the wrist had only recently subsided. In March, 1932, the tumor recurred, and another curettage was performed, followed by irradiation. On December 28, 1932, she was admitted to the French Hospital, where another curettage was performed for a sarcoma of the radius.

Microscopical report: "Mass of curettings from bone shows areas of degenerated tissue hemorrhage and mass of undifferentiated cellular connective tissue. Scattered through this latter tissue are many large giant cells of the epulis type. The tumor as a whole is only moderately vascular. *Microscopical diagnosis:* giant-cell tumor."

On April 13, 1933, the patient was readmitted to the French Hospital and another curettage was performed for a recurrent giant-cell tumor. *Microscopical report:* "Histological section shows a cellular mass of undifferentiated connective tissue cells, scattered through which are innumerable giant cells of epulis type. The tumor as a whole is extremely vascular because of cellularity of stroma. This type of giant cell would be classified as the aggressive type."

In the summer of 1933, the patient was under the care of Dr. A. Lightstone of New York. On August 18, 1933, *roentgen-ray examination* by Dr. J. Townsend Trevers, revealed: "The distal 2 inches of the radial shaft markedly widened and the presence of numerous multilocular cysts noted, extending downward to the distal articulating surface. There is a sclerotic

extend throughout the fatty tissue and into the underlying muscle. There are large blood vessels in some of the fibrous tissue strands. These show markedly thickened walls in some instances. The fibrous tissue and fat are diffusely infiltrated by lymphocytes, plasma cells and polynuclear leucocytes. In the deeper part the fibrous tissue and muscle are undergoing necrosis.

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"*Note:* The malignant characteristics of the neoplasm are not so marked as in the previous specimen."

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zone of reaction in the ulna and proximal margins, walling off the lesion. The external margin reveals a thin shell-like cortex. The dorsal aspect of the lower end of the radius is completely denuded (postoperative interference) and the tumor extends out into the soft structures along the dorsal and external regions of the lower forearm and wrist.

"*Conclusion:* The failure of bone to regenerate is due to the neoplasm extending into the soft structures, and is probably a virulent type of giant-cell tumor. Such long-standing neoplasms may undergo malignancy. It would be interesting to ascertain the result of the biopsy following operation at regression of new growth, the question of resection plus Coley's serum is to be considered. No evidence of metastatic involvement of the lungs or ribs."

The patient was admitted to the Sydenham Hospital in August, 1933, where she was operated upon by Dr. Lightstone on September 1, and again on November 25. The *microscopical diagnosis* on each occasion was: benign giant-cell tumor. From September 15 to December 15, 1933, further irradiation was given (1800 R., 50 per cent doses).

She was referred to me by Dr. Lightstone on December 8, 1933, at which time physical examination showed an old scar in the median part of the dorsal surface of the lower end of the forearm just above the wrist. On the inner side over the inner part of the radius was a large fungating tumor about the size of half an egg, from which was a very foul discharge. Recent roentgenogram showed a marked destruction of the lower end of the radius. There was marked limitation of motion at the wrist. An amputation was advised, but to this the patient would not consent. During the next four weeks she was treated with heavy doses of Coley's toxins, with only slight improvement. She finally consented to an amputation, and this was performed by me at the Hospital for Ruptured and Crippled.

Microscopical diagnosis: Malignant osteogenic sarcoma. This was confirmed by the Memorial Hospital laboratory. At the same time sections obtained the previous operations were examined and pronounced benign giant-cell tumor.

This case, I believe, is one of the most convincing of our series. The evidence is very strong that the original tumor was a typical benign giant-cell tumor controlled temporarily by irradiation. It is possible that the continued use of irradiation after curettage may have had some influence in bringing about the malignant transformation of the tumor.

CASE XI. *Benign giant-cell tumor of upper end of humerus (microscopical diagnosis, Memorial Hospital laboratory). Clinical and x-ray*

diagnosis: malignant osteogenic sarcoma. Amputation. Recurrence in scapula two and one-half years. Diagnosis: osteogenic sarcoma. Patient well five and one-half years after amputation.

J. McC., female, aged eighteen years, was admitted to the Memorial Hospital on July 25, 1929, with a history of having felt pain in the left shoulder immediately after an attack of influenza seven months previously. Physical examination on admission showed a symmetrical enlargement of the upper third of the left humerus associated with moderate tenderness and heat. A diagnosis of osteogenic sarcoma was made by Dr. William B. Coley, and Dr. Bradley L. Coley.

Radiographic report of Dr. Herendeen, July 25, 1929, was as follows: "Stereo views of the left shoulder reveal evidence of a destructive process involving the upper end of the humerus. It appears to be medullary in origin, but the growth has been so rapid that very little dilatation of the medullary portion has taken place, without any evidence of an attempt at limitation through callous production. The medullary portion of the shaft below this area is altered in density and it appears that this portion is involved also, although some of the loss of bone density may be due to atrophy of disuse.

"*Diagnosis:* Malignant primary medullary bone tumor. There is no evidence of metastases seen in the film of the lungs."

Dr. M. Q. Ewing of Armory, Minn. who referred the patient to Doctor Coley stated that there had been considerable doubt as to the diagnosis, some regarding the condition as chronic osteomyelitis and others as bone tumor.

In our opinion an amputation was strongly indicated, and while waiting for permission to proceed, the patient was given 50,000 mc. hr. of radium (emanation pack) between July 25 and August 7, 1929, and four doses of Coley's toxins ($\frac{1}{80}$ minim intravenously). On August 14, 1929, an amputation was performed by Dr. Bradley L. Coley.

Pathological report: "*Gross:* Tumor of the humerus involves the whole of upper extremity of bone, measures $7 \times 5.5 \times 4.5$ cm., has expanded and destroyed the bone, but is nevertheless completely encapsulated, with the exception of the point where the round ligament has been severed. Structure is cystic, multiple cavities filled with reddish fibrin and whiter opaque glistening interstitial areas. Tumor extends about 2 cm. along the shaft.

Diagnosis: Benign giant-cell tumor."

On April 18, 1932, or two and one-half years after the amputation, the patient was examined by a school physician, who found a growth about the size of an English walnut just below the clavicle in the shoulder-joint; this had been noticed by the patient for some time. An operation was per-

formed by Dr. M. Q. Ewing on May 5, 1932, and the specimen sent to the Memorial Hospital for examination.

Pathological report: "*Gross:* Left scapula. An old case of giant-cell tumor of upper end of humerus; recurrent mass nearly three years postoperative. There is a soft pinkish-grey recurrent tumor in the soft parts just below and posterior to the glenoid. Measures 3 cm. in diameter; is attached to the periosteum of the scapular spine but does not invade the bone. Can easily be freed from periosteum.

"*Microscopical:* Bulk of this shows giant-cell tumor; however, portions are lying down bone, other parts have lost giant cell and the tumor is malignant." (Osteogenic sarcoma.)

A letter from the family physician in November, 1934, stated that the patient was still well, more than five years after the amputation.

This case cannot be regarded as a benign giant-cell tumor which later became malignant, but is offered as an example of a microscopically pronounced benign giant-cell tumor which recurred two and one-half years after amputation, the recurrent tumor then being pronounced malignant. It is further worthy of note for the reason that Bloodgood, Geschickter and Copeland, have repeatedly stated that there is no case on record of a giant-cell tumor recurring after amputation. The clinical course in this case shows that the tumor must have been of a very low-grade malignancy.

CASE XII. *Osteitis fibrosa cystica apparently cured by irradiation; recurrence seven years later; further irradiation; amputation; microscopical diagnosis; malignant osteogenic sarcoma; metastases; death.*

M. J. N., female, aged thirteen years, was admitted to the Memorial Hospital, on the service of Dr. D. Quick, on April 7, 1919. The patient complained of pain and swelling in the tibia in 1919. The pain subsided but a small lump remained. This showed no tendency to increase in size until six months later when there was a similar attack of pain. Physical examination on admission showed an enlargement of the upper portion of the right tibia (9 × 5 inches); this, on roentgenological examination, was said to have the typical structure of osteitis fibrosa cystica. It was characterized by a cystic-like cavity formation, obliteration of medullary cavity and destruction of the cortex over the anterior surface of bone up to the limits of the periosteum.

Irradiation (radium and *x*-rays) was employed in the early part of 1919. The patient remained well until August, 1927, when there was evidence of

increased activity at the site of the old lesion. The radium-pack (14,000 mc. hr.) was applied on August 22, 1927, and again (12,000 mc. hr.) on November 15, with no appreciable improvement. In January, 1928, two months later, the clinical and roentgenological evidence of a malignant osteogenic sarcoma was so strong that an immediate amputation was advised and performed by Doctor Quick.

Microscopical diagnosis: osteogenic sarcoma; large polyhedral cells. In April, 1929 the patient developed pulmonary metastasis, and died in June, 1929.

This case is included for the reason that Bloodgood, Geschickter and Copeland regard osteitis fibrosa cystica as belonging to the same group as benign giant-cell tumors.

CASE XIII. *Giant-cell tumor of humerus; amputation; death from pulmonary metastases fifteen months later.*

H. B., male, aged ten years.

Trauma: Fell, fracturing humerus, shortly after which a rapidly growing tumor developed in the upper end of the humerus. In view of the malignant features of the tumor, an amputation was performed without a biopsy. *Microscopical diagnosis* by Dr. Ewing: giant-cell tumor of the epulis type. A brief postoperative course of Coley's toxins was given by the family physician. About one year later, the patient developed extensive pulmonary metastases, and died in a few months.

On reviewing the sections of this tumor, it was stated that the original microscopical diagnosis of *benign giant-cell tumor* was an error, and that the tumor was really a highly malignant osteogenic sarcoma. The fact remains that the histological structure of the tumor so closely resembled that of a giant-cell tumor that it was so regarded by a pathologist of great experience in bone tumors.

CASE XIV. *Giant-cell tumor of femur. Amputation. Death from metastases.*

E. R., female, aged sixteen years, had a tumor of the lower third of the femur of three months' duration. *Clinical diagnosis:* central sarcoma, malignant. *Microscopical diagnosis:* giant-cell sarcoma. The toxins were given for one month but in spite of this treatment the disease continued to progress, and an amputation was performed on May 18, 1906. The patient remained well for three years, and then died of metastasis to the pelvic bones, and probably to the lungs as well.

In this case there is little doubt that there was an error in the original diagnosis, and that the so-called giant cell tumor was a malignant osteogenic sarcoma from the start.

The following case is somewhat similar.

CASE XV. *Giant-cell and spindle-cell sarcoma of humerus. Amputation. Brief course of toxins. Metastases. Death.*

R. F., female, aged fourteen years, was admitted to the Memorial Hospital in August, 1911, with a rapidly growing tumor of the upper end of the humerus which had all the clinical signs of malignancy. I performed an immediate shoulder-joint amputation without a biopsy. *Microscopical diagnosis*, Dr. Ewing: giant-cell and spindle-cell sarcoma of epulis type. The tumor recurred locally, produced pulmonary metastasis, and ended in death on October 28, 1911.

This was a case in which the clinical evidence (rapid growth and appearance of tumor) made it possible to arrive at a positive diagnosis of osteogenic sarcoma without regard to the microscopical examination.

CASE XVI. *Benign giant-cell tumor of the tibia, which later became malignant.*

M. L., male, aged forty-four years, had a sudden onset of pain in the right leg just below the knee in 1926. The condition was diagnosed as a bone cyst, and an operation advised but refused by the patient. During the next few years occasional *x*-ray pictures were taken; one in 1929 showed what appeared to be a giant-cell tumor of the head of the tibia. A second similar attack of pain occurred in October, 1932. In August, 1933, he was admitted to Mt. Sinai Hospital where Dr. Neuhof performed a curettage with primary wound closure. *Microscopical diagnosis:* benign giant-cell tumor. These sections were reviewed by Dr. Stewart of the Memorial Hospital, who reported as follows: "The bulk of this lesion is typical benign giant-cell tumor. In one portion, however, the process is not typical. It shows peculiar thick-celled granulations vessels surrounded by sheaths of plasma cells and accompanied by a reticular cell layer resembling perivascular histiocytes. These gradually merge into spindle cells."

The pain persisted and on November 4, 1933, the patient was admitted to the Memorial Hospital on the service of Dr. Bradley L. Coley. *Clinical and roentgenological diagnosis:* giant-cell tumor. A second curettage was performed on November 11, 1933, and the wound closed without drainage.

Microscopical examination: "Most of this tissue is chronic productive fibrosing inflammation. One small focus shows a suggestion of giant-cell

tumor. In another area the peculiar overgrowth of perivascular cells duplicates that described in the first specimen. The process does not appear more active than did the first specimen."

At this time there was noticeable enlargement not only of the upper end of the tibia but apparently of the lower end as well. X-ray examination of the chest was negative. The pain persisted, and on January 3, 1934, the patient was admitted to the Hospital for Ruptured and Crippled.

Radiographic report by Dr. R. Lewis, January 3, 1934: "Examination of the right knee shows a destructive and productive lesion in the upper extremity of the tibia which probably started in cystic form. It is now irregularly destructive and the bone presents a moth-eaten appearance. The extreme destruction, the irregularity of the destruction and the moth-eaten appearance suggest that the growth is malignant. The joint is apparently more or less involved as there is much clouding and distention of the joint capsule. Examined of chest negative for metastasis."

On January 6, 1934, an amputation was performed by Dr. Bradley L. Coley.

Microscopical report by Dr. J. E. McWhorter: "Sections show a diffusely infiltrating mass of undifferentiated connective tissue cells that are of the short spindle type. Scattered throughout the section are many mitotic figures.

"*Diagnosis:* osteogenic sarcoma."

Although a high thigh amputation was performed, there was an area of induration surrounding the femoral vessels and extending 1½ inches above the limits of the amputation. This proved to be an embolic metastasis in the femoral vessels. After the amputation it was found that the sarcoma of the tibia had extended into the knee joint and involved the lower end of the femur. A cycle of high-voltage *x*-rays was given at the Memorial Hospital from January 25 to February 13, 1934; this was repeated between April 5, 1934 and April 21, 1934. On May 16, 1934, chest plates revealed definite pulmonary metastases; the patient's course was steadily downhill and he died on July 20, 1934.

Comment: In this case there was a long history of trouble over a period of six years. The condition was originally diagnosed as cyst of the upper end of the tibia. At the first curettage, six years ago, a microscopical diagnosis of *benign giant-cell tumor* was made. A very rapid recurrence followed, and a second curettage with the same diagnosis was made. A very rapid recurrence followed with early involvement of the femur. Microscopical examination after amputation showed

the tumor to be a malignant osteogenic sarcoma. In all probability the malignant changes had already taken place before the first curettage, and the curettage was not responsible for the malignant changes.

CASE XVII. *Giant-cell tumor of humerus. Amputation. Death from metastases.*

C. H., male, aged fourteen years; in 1913 developed a tumor in the humerus at the site of a recent fracture which had been united by Lane plates. The plates were removed six weeks later on account of infection. A few weeks later, an exploratory operation was performed.

Microscopical diagnosis: giant-cell sarcoma of the mixed type. One year later the patient was referred to me. He then had extensive metastases to the femur, the ilium, and probably the lungs. Death occurred a few months later.

CASE XVIII. *Giant-cell tumor of radius; resection. Recurrence in ulna treated with toxins. Amputation. Metastases; death.*

C. R., male, aged twenty-two years, first noticed a tumor in the lower end of the right radius in May, 1913. The pain and swelling gradually increased. *Roentgenological diagnosis:* benign giant-cell tumor. In September, 1913, Dr. Howard Lilienthal did a resection of the lower third of the radius. Eight months later the disease recurred in the ulna, and the patient was referred to me for toxin treatment. A spontaneous fracture had occurred. Only a short course (8 injections) was given. He later returned to Dr. Lilienthal who performed an amputation of the humerus through the lower third. Microscopical examination showed giant cells present. Pulmonary metastases developed, and the patient died in December, 1915.

CASE XIX. *Giant-cell tumor of the femur. Diagnosis based on clinical and roentgenological evidence confirmed by microscopical examination. Six months later proved to be osteogenic sarcoma.*

A. K., male, aged eighteen years, was admitted to the Hospital for Ruptured and Crippled on October 23, 1929. He gave a definite history of trauma: hurt his knee while diving two months previously. One week later he felt pain in the knee which increased in severity and one month later a swelling was noticed. A roentgenogram taken on October 18, 1929 was reported on by Dr. Raymond Lewis, as follows: "There is some irregular calcium deposits on this new tissue. The appearance does not suggest an osteogenic sarcoma, it does not seem characteristic of a giant-cell tumor, though it might actually be this latter. It might be a chondroma, but the bony surfaces seem somewhat too irregular to really suggest this; it might be a myeloma." Another roentgen-ray examination made by

Dr. Harzmark on October 19, was reported as follows: Examination of *x*-ray plates made shows that the internal condyle of the femur is almost entirely replaced by a new growth which seems to be of the rapidly growing type. The upper aspect of the condyle is almost entirely obliterated. Small calcareous deposits may be seen within the bone and a more or less lacy filamentous network seems to be growing out from the edges. The patient's general condition shows a loss of weight. Diagnosis: neoplasm.

Physical examination on admission showed enlargement of the inner condyle of the left femur associated with slight limitation of flexion; extension normal; no joint involvement. The roentgenogram seemed to show a condition of osteitis fibrosa or giant-cell sarcoma which had involved almost the entire inner condyle so that the cortex was hardly discernible. On October 28, 1929, an operation, under an Esmarch bandage, was performed by Dr. Royal Whitman. "The mass was found to be very elastic, evidently on the verge of breaking through. On incision this was found to consist of only small fragments of eroded bone; beneath it there was a large grayish mass of tumor, the size of a lemon. This was removed entirely. The cavity was thoroughly scraped, swabbed out with pure carbolic acid and alcohol, and a flap of the vastus internus was cut and forced as far as possible into the cavity."

Microscopical diagnosis, Dr. W. H. Probert, Pathologist of the Hospital for Ruptured and Crippled: giant-cell tumor.

A roentgenogram taken on November 9, showed a very satisfactory result. By November 23, the patient was free of pain and able to walk about. A roentgenogram taken on January 22, 1930, showed no evidence of any increased destruction of bone although there was moderate atrophy of the shaft in the supracondylar area. Dr. Kleinberg believed the swelling of the soft parts to be due, probably, to a hematoma. The patient was discharged wearing a plaster knee bandage, and was requested to return in about one month for observation. Physiotherapy was given under the direction of Dr. K. G. Hanssen until March 8. No improvement followed but a steady increase in size was noticed. On March 11, the patient was examined by Dr. W. B. Coley who made a definite clinical diagnosis of osteogenic sarcoma, which he believed was confirmed by new roentgenograms taken at that time as well as by previous ones just examined by him. He advised an immediate amputation.

Roentgen-ray report by Dr. R. Lewis, March 10, 1930: "Anteroposterior and lateral views of the left knee compared with the last films taken in January, 1930 show considerable more bone destruction and a much larger soft tissue mass, containing within it irregular calcifications. The condition is undoubtedly an active malignant tumor."

An amputation was performed by Dr. W. B. Coley on March 19, 1930.

Microscopical examination by Dr. J. E. McWhorter: "Sections show for the most part an exceedingly cellular mass of undifferentiated connective-tissue cells somewhat of the spindle type. These cells have a highly chromatic nucleus and each high-power field shows many mitotic figures. Diffusely scattered throughout this cellular mass are many moderately large giant cells of the tumor type. These average from four to six nuclei. The tumor as a whole is very vascular with blood sinuses lined by tumor cells. No new bone seen in sections. Tumor is evidently of osteogenic type. *Diagnosis:* osteogenic sarcoma."

Treatment with Coley's toxins was begun on April 2, 1930; most of the next 16 injections were made intravenously. The patient was discharged on May 4, 1930.

He was readmitted in the following month and given further toxin treatment. This was kept up with occasional intervals of rest for the next few months. A roentgenogram taken in November, 1930, showed evidence of lung metastasis. In December, 1930, the radium pack was applied. The disease continued to progress, and on April 1, 1931, the patient died. A revision of the original sections showed that there was, undoubtedly, an error in the diagnosis of giant-cell tumor, and that it was an osteogenic sarcoma from the start.

CONCLUSIONS

1. I believe that later observations have confirmed the conclusion reached in my paper of 1924,[20] viz: "In view of the cases here reported, it would seem necessary to modify the opinion so strongly held by most of the leading pathologists of today, that giant-cell sarcoma is always benign and never gives rise to metastases."

2. While some of the cases reported as benign giant-cell tumors that later become malignant, may be best explained as *errors in diagnosis*, there is a considerable number in which the evidence is very strong that the primary tumor was a true benign giant-cell tumor which later, due to some unknown cause, changed in character to a malignant metastasing tumor. This distinction is really only academic and of little practical moment to the surgeon or the patient.

3. The percentage of cases in which tumors diagnosed as *benign giant-cell tumor* by experienced pathologists have later

become malignant, although small, is actually much larger than has been recognized by recent writers. It is probably in the neighborhood of 15 per cent.

4. The acknowledgment of the fact that a considerable number of giant-cell tumors do later become malignant should not lead us to abandon conservative methods of treatment for more radical measures (amputation). It should increase our efforts to arrive at a correct diagnosis at the earliest possible moment. Furthermore it should lead us to caution the patient with an apparently cured giant-cell tumor to avoid any local trauma.

5. The fact that such a high percentage of our series of malignant giant-cell tumors were treated by irradiation (radium or *x*-rays) after curettage suggests that this mode of treatment should be abandoned. If irradiation is the method of choice, it should be used alone and not in conjunction with curettage.

6. My experience up to the present time forces me to conclude that surgery (curettage, chemical cauterization, without packing or drainage) with or without postoperative toxin treatment, offers the greatest percentage of permanent cures with the shortest period of disability and the least impairment of function. A brief period of postoperative toxin-treatment (four to six weeks) will, I believe, materially lessen the number of recurrences.

7. The fact that such a considerable number of cases diagnosed as benign giant-cell tumor have later become malignant leads one to ask whether we should continue to use the term *benign giant-cell tumor* or whether we should return to the older classification of *giant-cell sarcoma*.

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THE ADDED PROBLEMS PRESENTED BY

GAS GANGRENE AND TETANUS IN COMPOUND FRACTURES

RALPH G. CAROTHERS, M.D., F.A.C.S.

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FRACTURES which are compound are more difficult to treat and require greater skill in splinting than do simple fractures. The compound fracture which is infected with anaerobes, and in which either gas gangrene or tetanus develops, presents one of the most complicated problems in surgery. Reports of 2 such cases, including more or less detailed accounts of the complications which developed are, we believe, as good a way as any to bring this subject to your attention. The first of these 2 cases was the first time we had seen these two diseases in combination. It is not strange, therefore, that many times we wished we had done this or that differently. When the second case came under our care a few months later, we were able to carry out these ideas with what success the case report will record.

CASE 1. A young man, freshman in college, was driving a car which was wrecked. The circumstances of the accident, which were not known by us for several days, were important and were as follows: The car, while proceeding slowly through a sunken cut in a country road, skidded into the ditch on the left side and overturned against the bank. The driver put his left arm through the window as the car went over, and presently found his arm pinned between the frame of the door and the earth. Both bones of the forearm were fractured at the junction of the middle and lower thirds, and the lower ends of the upper fragments were each compounded through small seperate lacerations on the flexor side of the forearm. The patient dug the earth with both hands, pulled and struggled considerably and finally was able to get his arm loose. We saw him about one hour after the accident, but not before he had been undressed and given a narcotic. Therefore we did not know that there had been considerable trauma to the forearm above the site of the fracture, and we did not know that the patient

had used the arm considerably and violently after the accident. As we saw the case, it was one of fracture by indirect violence, compounded from within out, and relatively clean. Excision of the wounds, reduction of the fractures, and closure were not difficult. The reduction was so good, the case seemed such a clean one, and the treatment rendered so early, that we were led immediately to apply a plaster cast. The patient was given a dose of mixed tetanus and gas gangrene antitoxin.

On the second day gas gangrene developed. The cast, which had already been split on one side was now bivalved, and the flexor half removed entirely. Long incisions were made, all muscle obviously necrotic excised, and large doses of antitoxin started both intravenously and intramuscularly. It was believed that the inflammation was confined to the flexor side of the forearm, and it was hoped that, with the arm resting in the posterior half of the cast, a reasonable reduction might be maintained. Indeed such seemed to be the promised outcome for with 160,000 units of antitoxin the inflammation gradually subsided, and it was possible to apply the cast again, leaving a window for dressing. During all this time a 1:5000 aqueous solution of merthiolate had been used in the wounds.

On the tenth day a rather severe case of serum sickness developed, and with it some bizarre symptoms of paralysis of muscles of ocular control. This last was believed to be only one more expression of serum reaction. After a few days the skin eruptions were subsiding but the eye muscles were even more affected, and there was some difficulty in swallowing. At this time twiching of the muscles of the left arm began to occur. This was thought to be caused by the irritation of badly splinted fractures, for by this time, the cast was loose and not fitting well. A new cast was applied and the arm twichings were less noticeable for a few days but soon began again in real earnest. It was then that we became aware of the fact that we were dealing with a case of tetanus of the local-paralytic type. That is, the contractures were confined to the injured limb, and there was paralysis of some muscles.

At this stage the patient was so sensitive to horse serum that 0.1 c.c. of a 1:10,000 dilution gave a marked reaction. One interne was relieved of all other duties, and beginning with a still smaller dose, and injecting every fifteen minutes, was able after fifty-six hours to get the patient in such condition that he could take ordinary doses of tetanus antitoxin. With the first full dose of serum, the paralysis of the eye and throat muscles began to clear up, and in a short time they were functioning normally. However, during the period between the time when the diagnosis of tetanus was made and the full doses of antitoxin were given, the disease had progressed from one of local, to ordinary generalized tetanus. The cast on the arm had again become loosened as the edema of the previous inflam-

mation subsided, and during the severe general contractures not only was the cast itself nearly broken but the patient did recompound his fragments through the posterior surface of the forearm. In spite of all of this full strength doses of antitoxin soon became effective. One hundred and sixty thousand units were used with an encouraging result from the start. There were two rather severe complications, i.e. myocarditis and hemorrhagic nephritis, which kept the patient in bed for about six months, but these also cleared up, and he was able to return to college again in the fall.

As to the arm; the inflammation subsided almost entirely. Two pieces of bone each about 4 cm. long and each sloughing from the lower end of the upper fragment of each bone, came away. The arm shortened and united in this new position with some angulation. It is not a pretty arm, but it is alive and it functions well enough for the patient to shave and dress himself and to drive a car, and function is improving. The saving of this arm cost the patient six months in the hospital and another five months away from his work. We believe however that it has been worth while.

At the same time we hoped that some of the complications might, in a future case, be avoided, and a better result obtained more quickly. With this end in view we asked ourselves the following questions:

(1) Can gas gangrene or tetanus be anticipated with any degree of accuracy, and if so, what steps might be taken to prevent the disease or lessen its severity? (2) Should a compound fracture be excised and closed if an anaerobic infection is suspected? (3) Should plaster-of-Paris be used in the early treatment of such cases? (4) Does tetanus usually go through a stage of local tetanus before generalized tetanus develops?

In going over the histories of our cases of gas gangrene seen since army days, we found rather similar circumstances in all cases, save one. This latter was a case of gas gangrene developing in the abdominal wall following a operation for obstructed, but not strangulated umbilical hernia. The other 10 cases gave such histories as these: "sliding in cow pasture, tore leg open," "run over by hand car at a rural railroad crossing," "broke arm in barn yard," "fell out of a tree in an orchard," "broke arm in the barn," "broke leg in recently fertilized park," "broke arm in cellar where there was tan bark floor," "broke arm on road side next to a pasture," and

two other cases of compound fracture occurring in a school yard from which other cases of gas gangrene had been known to develop. Thus, in all cases except the hernia case and the school yard cases, there was a direct connection with cattle or horses or animal products such as fertilizer or tan bark. In addition one or both of the following factors was present in each of these cases, *i.e.* the bone ends had been driven into the soil, there had been a severe crushing or laceration of muscle tissue.

The cases of tetanus were found to have similar histories which were as follows: "rusty nail in a hen house," "scratched hand on license plate of car which was kept in cow shed," "shot in foot with blank, the wad not removed," "broke arm on road next to pasture," and "broke arm in cellar where there was tan bark floor."

Therefore, it seems that both gas gangrene and tetanus might be suspected in cases occurring in certain localities, and to some extent anticipated. As a matter of fact we did, in the cases of this paper, anticipate both diseases to the extent that we gave prophylactic doses of serum for each. But, as we discovered, one dose of serum is not always a preventative, and if one is really to anticipate a disease, he must be prepared to treat it in the best way, should his plans for prophylaxis go astray.

An arm or leg, already the seat of a compound fracture, in which gas gangrene develops, can, we believe, be treated far better when splinted by the double pin method, as described by Griswold, Caldwell and others, than by any other we know. Or if tetanus develops, this type of splinting is the best, and we determined therefore, that, when the next opportunity presented itself, that is when next we felt, that in spite of all we could do in the way of prophylaxis, either gas gangrene or tetanus might develop, we would insert a pin through the fragments well below the fracture and if possible another well above, and lock these pins to the bars of a Thomas splint. Fragments so splinted, we believed, would remain splinted

through any calamity, and at the same time permit any other surgical procedure.

But to return to the matter of prophylaxis, it might properly be asked whether early closure of the wounds is justified in these cases. Of course, if one knew for certain that severe infection was about to develop, he would not close the wound, but one never knows. We believe that excision and immediate or delayed closure, depending on the extent to which the surgeon is able to excise, and the time at which the case is seen, is a firmly established surgical principle. Too many dirty looking cases have been successfully excised and closed, and the cases which have had to be reopened were no worse off for the procedure. As a matter of fact in our experience, gas gangrene has developed as often in cases which were not closed, as it has in those which were.

Serum: No large series of cases of gas gangrene treated with serum is available, nor has the serum been used prophylactically for very long. In our small series however, the serum seems to have been of great value. One patient died of gas gangrene of the abdominal wall, having received no serum. This was our only death, but in 4 other cases treated without serum, amputations were necessary. In 5 additional cases, treated with serum both prophylactically and therapeutically, amputations were not necessary, and in one other treated with heavy therapeutic doses, a leg was saved. The prophylactic dose of antitoxin seems to have delayed the onset of the disease, when it did develop, and to have lessened the toxicity, and of course one will never know how many, if any, cases were entirely prevented by this procedure.

Therapeutically we have given the serum in large doses, i.e. 40,000 to 60,000 units a day, both intravenously and intramuscularly. The surgical procedures in addition have been simple, and have consisted merely of splitting fascia on one or both sides of the limb for a generous length. Thus even severe edema could not strangulate the circulation. Gas gangrene kills locally, by developing such an edema in a sleeve of fascia

that circulation is impossible. It might be likened to an internal expanding brake. And it kills generally, that is the body as a whole, by toxemia. Therefore, the treatment should be along lines similar to that of diphtheria, where we keep the air passages open, surgically if necessary, and cure the disease with antitoxin.

In the matter of tetanus, we believe that an original excision and the prophylactic dose of antitoxin will prevent development in most, in fact nearly all, instances. However, a patient who develops gas gangrene must surely have been infected with tetanus bacilli also, and the reason the two diseases have not been seen in combination more often must be the effectiveness of the original 1500 units of A. T. S. most of these patients received. One dose of serum does not make it absolutely certain that tetanus will not develop, but it does delay the onset of the disease and in the majority of cases absolutely prevents. Only a few cases of gas gangrene both keep their limbs and at the same time live long enough to get tetanus. Millar in reporting 607 collected cases of gas gangrene in civil life, found that half had died, and another 20 per cent had had amputations, leaving only 30 per cent to get tetanus at all, unless it developed quite early. While we had not seen tetanus develop in a patient receiving a prophylactic dose of serum, we determined that in the future, we would give, in any case of gas gangrene, multiple doses of A. T. S., and do this while the gas gangrene serum was being administered, so as to get it in before the patient became sensitive.

It was not long before we had an opportunity to try out these ideas.

In May of this year a young woman came to the hospital suffering from compound fractures of both bones of the right forearm. The accident, which had occurred about an hour before we saw her, was interesting and similar in essentials to that of the first case. This patient was helping guide a barrel of beer down into a cellar, when the barrel got loose, knocked her down, and ran up on her outstretched arm. The arm was palm down on the tan bark floor and the lower ends of the upper fragments were driven into

the tan bark having emerged from the forearm through two small holes on the flexor side, lower third. There was also the history that the barrel had run up on the arm as high as the elbow at least, thereby possibly bruising the bellies of the extensor muscles.

Physical examination did not reveal any injury above the fracture however. Operation was performed at once. The usual excision was done and the wounds closed. A pin was put through the olecranon, and another through the lower ends of the radius and ulna. These pins were then locked to the bars of a short Thomas splint and the patient put to bed with the arm elevated, that is the forearm perpendicular. Light dry dressings were placed on the wounds. The patient had already received 1500 units of A. T. S. before we saw her, and as we wanted her to have the 2000 units anti-gas gangrene prophylactic, she got an additional 1500 units of A. T. S. which was in this tube.

Twelve hours later there was no sign of infection, but in twenty-four hours there was some edema of the arm and some serus drainage from between the stitches. Gas could not be felt for certain, but a smear was positive and the second operation was performed at once. The patient was obviously toxic. Long incisions were made in the forearm front and back, the incisions including the fascia for the whole length. Foul-smelling dirty brown fluid came from the arm, the bulk of this putrid material seeming to come from the extensor side, and at least 4 inches proximal to the level of the compounding, in other words from the area of the bellies of the extensors. Also at this level, deep in the arm, gas was encountered. Dakin tubes were inserted and 1:5000 merthiolate aqueous solution used copiously. Serum was given in large quantities, 40,000 units a day being used at first and 180,000 units in all. By the end of the fourth day, which was the third day after the onset of the infection, the patient was decidedly better. We remarked to the interne that he must not forget to give another 1500 units of A. T. S. at the end of a week, but next morning, four and one-half days following the accident, tetanus began to assert itself. That is, there were twichings of the arm of just the sort the other patient had had for several days before the condition became generalized. We believed the condition to be tetanus and gave 40,000 units of A. T. S., half in the vein and half in the muscle, that same day. A culture was taken at the same time, and in short order tetanus and B. welchii were grown. In all, this patient received 50,000 units of A. T. S. in two days, the symptoms lessening from within a few hours after the first dose.

At the end of five weeks the arm was put into a cast with windows and in a few more days the patient went home to return as an out-patient. At the end of four months the wounds were well healed and there was fair function of the hand but no union of the fractures. In fact, just as in the

other case, about 1½ inches of the lower ends of the upper fragments had come away, and at the present there is considerable bony gap. This will have to be bridged by bone graft later.

CONCLUSIONS

The problem presented by gas gangrene and tetanus in compound fractures is splinting. We believe that when either of these diseases can be anticipated, the double pin method of splinting is superior to any other.

It appears that the prophylactic injection of serum is of value in gas gangrene, as well as in tetanus.

The rule laid down in the army that tetanus antitoxin be given weekly for a month, and not just once, should be remembered in gas gangrene cases.

DISCUSSION

Dr. Robert L. Rhodes, Augusta, Ga.: I think with these compound fractures that the keynote is the prevention of infection by the application of the principles laid down under the so called Carrel-Dakin treatment: thorough mechanical and surgical cleansing (debridement) followed by chemical sterilization. Dr. Carothers did not discuss in detail this phase of the subject. My feeling is that a fracture with any skin laceration, even a pinhead puncture, is a compound fracture and should be treated as such. The size of the skin injury is no index as to the extent of the damage within. If we scrub our hands, which are reasonably clean, for ten minutes in preparation for any operation, how much more urgent that we scrub ten, fifteen, twenty minutes these dirty compound injuries, many of which have been ground into the soil or clothing. Scrub as we go, layer by layer until we are sure that we have cleansed the skin and wound in all its pockets or recesses with particular attention to the torn, loose, reflected periosteum and the medullary cavities, in which often we will find foreign particles. My next step is to repeatedly flush the wound with ether, sprayed into all recesses with a bulb syringe, working from within outwards; it is an excellent cleansing agent, removing the soap as well, actively germicidal, quickly evaporates and aids very much in the recognition of normal tissue which bleeds readily and differentiation of devitalized tissue. Next the surgical cleansing is done by removal of all shreds and other devitalized tissue (debridement). Again the wound is thoroughly flushed with ether. Now if these steps have been done thoroughly, one may treat the fracture as in a clean operative case and close the wound tight, because we have removed

all foreign bodies and bacteria during the stage of contamination and before infection has taken place. If there is any question as to the thoroughness of these measures or if twelve hours, the maximum limit of time, have elapsed since the injury occurred, Carrel tubes are placed for irrigation and the wound either left wide open or only partially closed. Dakin's solution may be used instead of ether but it is troublesome to prepare, unstable and does not help me so much as ether in the differentiation of normal and devitalized tissue. I have had no experience with merthiolate but the fact that it is a mercurial and therefore corrosive would prejudice me against its use in such cases, as is true of all other corrosives. Where we have gone to such efforts to remove all foreign matter and devitalized tissue why immediately undo these efforts by producing an excellent culture medium in the form of a coagulative necrosis by the introduction of a corrosive chemical?

I recently had an experience that shows how the extent of the skin injury does not show the gravity of the situation within. A little child fell out of a tree and sustained compound fractures of both femurs, mid third. The left thigh was considerably lacerated and the wound well open, but the findings inside were much less severe than the right. The right showed only a little puncture wound ¼ inch in diameter, but I found the muscles lacerated, the periosteum stripped back 2 inches and in the medullary cavity of the upper fragment was a little plug of dirt. Had we not gone in and thoroughly cleaned out the wound, with the emphasis on the "thoroughly," we would not have found that dirt, and a long drawn out, severe infection would have resulted. Just as was said, we should take care of these fractures *immediately*. Our belief is that compound fracture is more an emergency operation than is an acute appendicitis. The peritoneum will take care of a whole lot more than the bone will.

Dr. W. Barnett Owen, Louisville, Ky.: My interest is always stimulated in any surgeon when he reports some bad results. After all, that is where we learn the most, not from 100 per cent cures. When one practices surgery a number of years he is certain to have his share of complications and failures.

My interest in skeletal fixed traction in the treatment of selected fractures has been greatly increased in the past few years. Lorenz Boehler, of Austria, appeared before a number of societies in this country several years ago, outlined his method of skeletal traction. Since that time a number of surgeons have become intensely interested. This method is of the greatest practical value in compound, comminuted fractures of the leg and arm, as reduction and maintenance of reduction can be continued as long as necessary, in the simplest way, without coming in contact with the broken fragments, and at the same time allowing free access of the

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leg for dressings and the application of heat and massage. It also makes it possible to permit the establishment of early use, without the fear of disturbing the broken fragments. It shortens hospitalization tremendously. It also prevents a great amount of bony atrophy which results from prolonged rest in bed without use of the limb.

The methods employed by Dr. Carothers are very similar to those of Dr. Caldwell, of Cincinnati, and Dr. Griswold, of Louisville. All of these men are doing most commendable work in the treatment of complicated fractures.

DR. RALPH G. CAROTHERS (*Closing*): We have used merthiolate because we have been led to believe that gas gangrene germs can swim around in Dakin's solution and like it. As a tetanus prophylactic they started in the war with 500 units but this was not sufficient. After giving it twice a week apart there was still trouble. After three doses there were fewer cases and after four still fewer, but there were still some. The use of 1500 units is an effort to get back to the one shot dose. I think if it is just routine, all right, that will do, but if the man has been in a position where he is likely to get infection he must have another shot. The 1500 units will inhibit the germs that are there then, but we must remember that another group is coming on from the spores and precautions should be taken against that occurrence.

OVARIAN THYROID

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IN a fairly critical review of the literature I have found less than 80 cases of struma ovarii reported up to the present time. It is probable, however, that the condition occurs more frequently than such reports would indicate. It is my desire to place on record an additional case at this time.

Pick found thyroid tissue in 6 out of 21 dermoids examined; Rohdenbrug found it 9 times in a series of 500 ovarian tumors (61 of which were teratomata) and King reports a similar finding 3 times in 500 ovarian tumors. Were it possible to have every ovarian tumor carefully analyzed to determine the presence of thyroid tissue the result would probably be very surprising as to its frequency.

The case I am reporting is the largest tumor on record so far as I am able to determine, measuring, as it does, 18 × 14 × 11 cm. and weighing 3¾ lbs. There was no evidence of other teratomatous elements in the cyst except thyroid tissue. Adult and fetal thyroid tissue were both present in the same specimen. There was no ascites. The patient had multiple nodules in what appeared to be an adenomatous goiter in her neck. Clinically, it was non-toxic and the metabolic rate was −8.

A great deal has been written on the subject of ovarian thyroid, much of which is controversial in nature, especially regarding the genesis of the condition. Aside from the difficulties of determining the frequency of such tumors, there seems to be some question about all of them being true cases of struma ovarii. Bauer, for instance, concludes from his case that most of them are really pseudomucinous cystadenomata and not teratomata. (It should be noted that pseudomucinous cystadenomata are probably teratomata.) Other writers are

convinced that all ovarian goiters are true teratomata. Aberrant thyroid nodules have been found many times in the neck, at the base of the tongue and even in the thorax, but

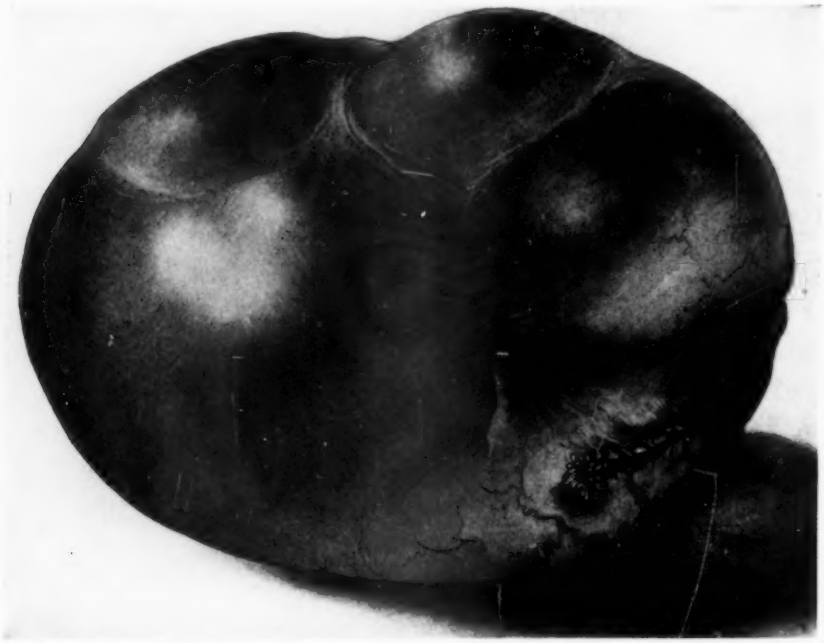

FIG. 1. Left ovarian cyst. Measurements, 18 × 14 × 11 cm. Weight, 3¾ lbs. Nodular mass is thyroid tissue.

isolated nodules of thyroid tissue found in an ovary certainly suggests the teratomatous origin of the growth. In the true dermoid type of ovarian tumor other structures, such as bone, teeth, hair, muscle and nervous tissue are often found. When the thyroid tissue predominates it would seem reasonable to believe that the endodermal elements of the thyroid anlage had undergone excessive development in the teratoma and had partially or completely suppressed the other tissues. Such a condition probably obtained in the case I am reporting, since no other elements of a true teratoma were found.

Frankl observed poor vascularization of the ovarian thyroid tissue and to this fact he attributed poor absorption of the colloid, accounting for the absence of hyperthyroitic symptoms. On the contrary, Kovac found adequate blood

supply in his case and he observed the subsidence of toxic symptoms following extirpation of the struma ovarii. Moench reports a similar case where the cardiac manifestations of

FIG. 2. Thin walled ovarian cyst open. The two nodular masses near the center are firm, encapsulated structures, composed entirely of thyroid tissue.

hyperthyroidism disappeared after removing an ovarian goiter.

Masson has recently written an extensive review of the literature and reports 6 additional cases from the Mayo Clinic. In some instances, he found evidence of hyperplasia in the ovarian struma and states that clinical evidence of toxic symptoms was present in some of them. He suggests that ovarian goiters may secrete thyroxin.

It was Pick who first stated that all cases of struma ovarii were really teratomatous in origin. He suggested the possibility

of an embryogenic disturbance, an embryonic displacement into the ovary of multipotent cells, which might in later life proliferate into the formation of thyroid tissue. The large

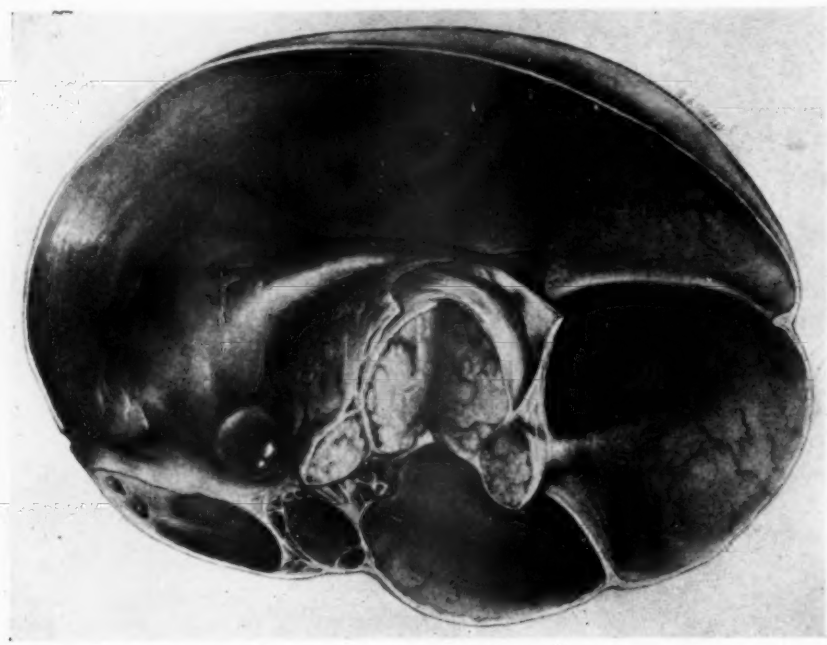

FIG. 3. Ovarian cyst open. Near center are two solid masses measuring 4 and 2.5 cm. respectively. They are completely separated from the ovarian tissue. The masses are identical and each contains fetal and adult thyroid tissue. No other dermoid elements found in the tumor.

majority of current writers are in accord with his views.

Macloed reports 2 cases of struma ovarii and agrees with Stevens that the term thyro-dermoid is a better term than struma ovarii for the reason that it more nearly describes the condition. It is believed by some men that pseudomucinous cystadenomata develop in a similar manner; that is, by a preponderant growth of the intestinal epithelial elements of an ovarian teratoma.

Shapiro reports a very interesting case of thyroid nodules found in the great omentum. He believes they were secondary to peritoneal metastases from a benign embryonically misplaced ectopic thyroid found in the ovary. He suggests the

parallel between this case and metastasizing pseudomucinous cystadenomata, which develop from the preponderant elements of intestinal epithelium, developing in ovarian teratomata.

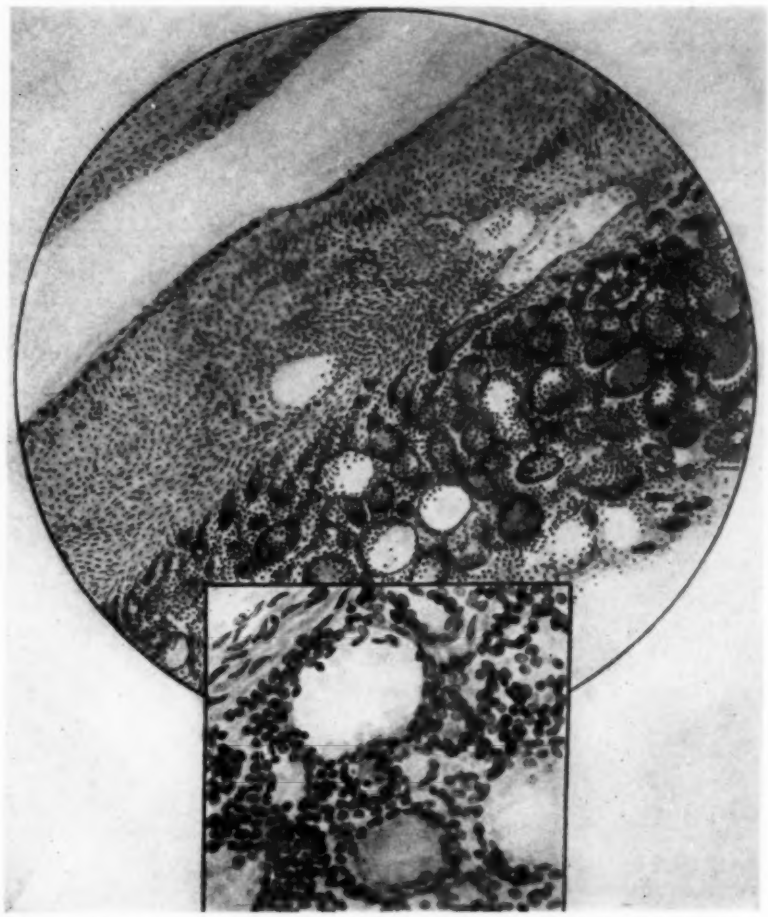

FIG. 4. High and low power sections showing thyroid tissue of adult and fetal type. Much colloid in some acini that are lined with single layer of cells. Proliferation and hyperplasia noted in other areas.

Thorough autopsy study convinced Shapiro that his case was not carcinomatous metastases. The struma colli was benign. The omental nodules did not suggest struma proliferans of the Langhans type.

After a very interesting series of experiments, Plaut is convinced that the thyroid tissue of an ovarian struma,

chemically, pharmacologically and biologically, behaves like true thyroid tissue. In some of his cases he found an ample supply of iodine in the ovarian goiter and, after feeding it to

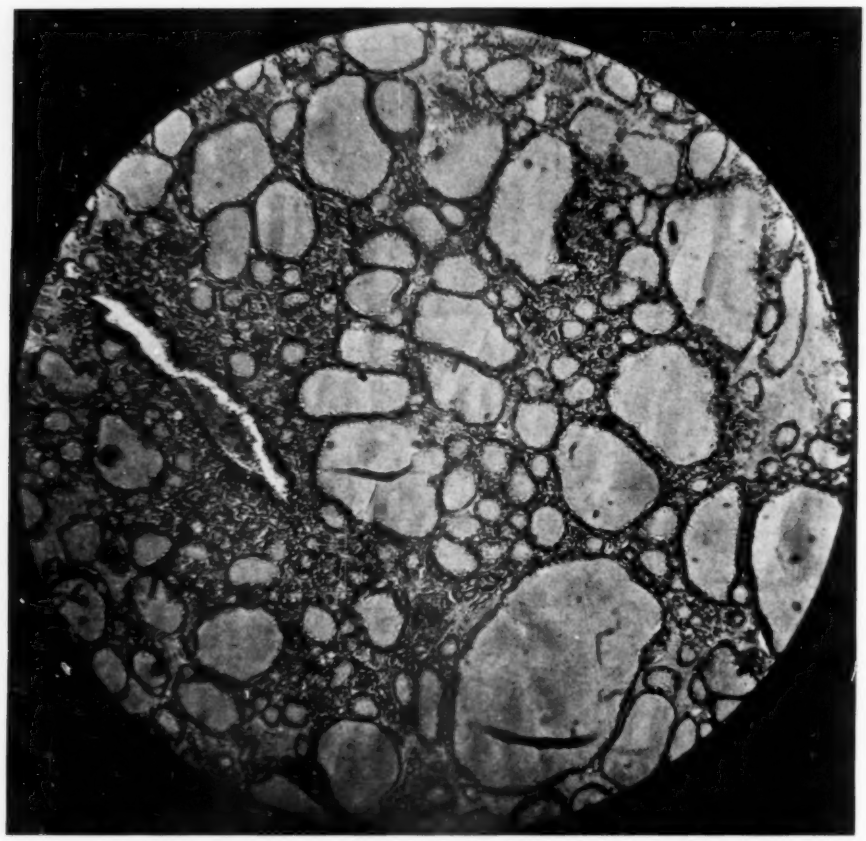

FIG. 5. Section shows separate, rounded spaces, supported by connective tissue stroma and containing homogeneous material (colloid). Acini lined by single layer of granular epithelial cells containing deeply colored nuclei. In the dense supporting stroma are masses of undifferentiated cells, probably young acini. The equality in size of acini, the colloid content and slight development of connective tissue, indicates normal thyroid elements in an ovarian cyst.

tadpoles and mice, the result was carefully noted. Growth and development were quickened and protection against certain poisons was developed in a convincing manner.

It would seem from the foregoing that we have gone far enough in our studies to be convinced of the true nature of

thyroid tissue found in struma ovarii. The condition is most likely teratomatous in origin.

CASE REPORT

Mrs. P. E. T., white, aged fifty-one, multipara. Except for diseases of childhood and a mild attack of influenza six months ago she has always been quite well.

Menstrual History: Onset at thirteen, regular except during gestation and lactation. Two years ago noted a slight bloody discharge at irregular intervals for one year. Ten months ago flowed freely for several days; none since.

Present Complaint: During the past eighteen months she has been aware of an abdominal enlargement, which has gradually increased in size. No pain or tenderness. There is a considerable feeling of heaviness in the lower abdomen, some pain in the back and down the thighs. A moderate amount of leucorrheal discharge that is not foul and non-irritating. She has been aware of a small goiter, especially on the right side of her neck, during the past four or five years. Choking, voice changes, nervousness, cardiac symptoms, changes in sight and other thyroid symptoms are absent.

Physical Examination: She is well developed, nourished and apparently in good health, 5 feet, 8 inches in height and weighs 160 lbs., which is her normal weight. There is no tremor, eyes normal, teeth in poor condition, tonsils not diseased.

Thyroid: A small nodular goiter is present, with the largest tumor about 1 × 2 inches, occupying the right lower lobe; other smaller nodules found on the left side. There is no thrill or bruit present. Lungs and heart negative. Blood pressure 120/80. Pulse 80.

Abdomen: There is a smooth rounded mass, filling the lower abdomen, extending to the level of the umbilicus.

Pelvis: Perineum moderately lacerated with poor support. Some mucopurulent material in the vagina. Small cervical polyp protruding from the external os, which is moderately lacerated and hypertrophied. The uterine fundus is slightly enlarged, pushed to the right by a large cystic mass that fills the pelvis and extends far up into the abdomen. Tumor can easily be separated from the uterus. It is thought to be an ovarian cyst, involving the left side. The urinalysis is negative.

Blood: W.B.C. 8600; hemoglobin, 80 per cent; R.B.C., 4,500,000. Blood sugar 100 mg. per 100 c.c. of blood.

Basal Metabolic Rate: Minus 8.

Diagnosis: (1) Dental sepsis. (2) Goiter, nodular, non-toxic. (3) Large ovarian cyst. (4) Lacerated perineum and cervix with cervical polyp.

Disposition: Operation for the removal of the ovarian cyst advised and accepted. The goiter and dental infection to be treated later.

Operation: May 30, 1933 at the Baptist Memorial Hospital, under

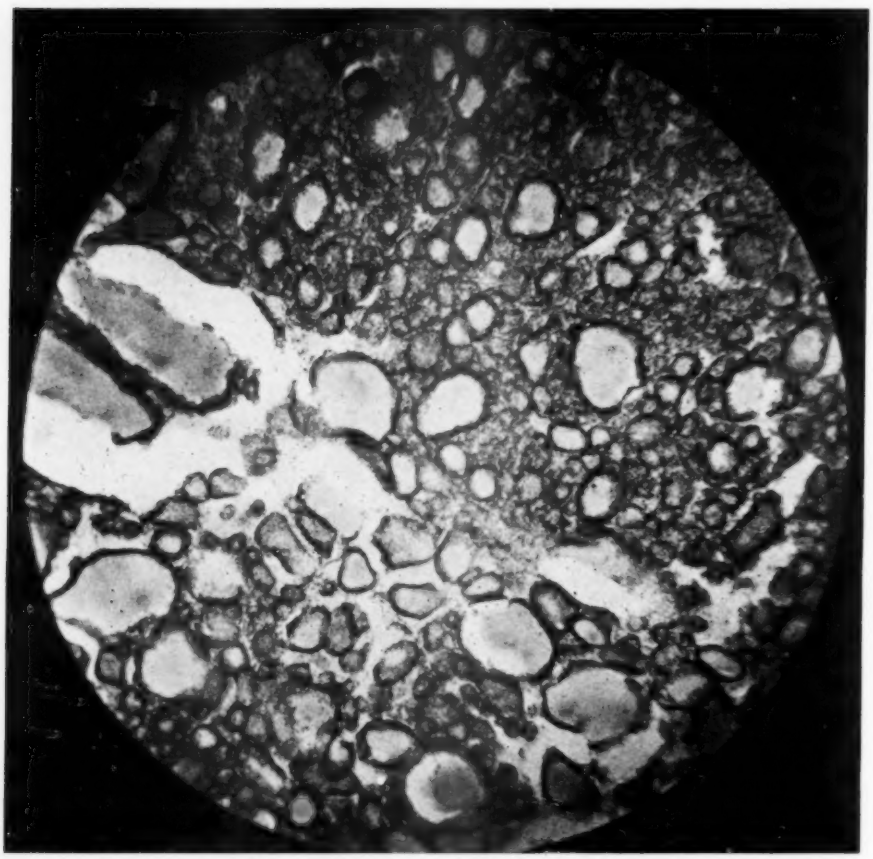

FIG. 6. Section from ovarian tumor. The vesicles are regular in size, some contain a little colloid, others none. In some areas the acini are closely packed. Between glandular acini are many cell masses without central lumen or cavity, similar in appearance to fetal or fetal adenomatous thyroid tissue. The apparent hyperplasia is probably due to increase in number rather than size of acini.

ethylene anesthesia, a long low midline incision was made and a total hysterectomy, a bilateral salpingo-oophorectomy and an appendectomy were done.

Remarks: No ascites. A large, smooth, multilocular left ovarian cyst was removed unruptured. The right ovary and tube were essentially normal in size but were atrophic. The uterus was increased in size, contained one fibroid. The cervix was elongated, lacerated and contained a polyp. The appendix was chronically diseased and was removed. There

were no gallstones and the exploration was otherwise entirely negative. No evidence of metastases. The wound was closed without drainage. Patient left the operating table in good condition. Postoperative convalescence satisfactory. Wound healed by primary union and she was dismissed from the hospital in two weeks. She has been seen several times since then and her health is quite good to now. A metabolic rate two months postoperative was still −8.

Pathological Report: Dr. N. E. Leake, pathologist at the Baptist Memorial Hospital, Memphis, Tennessee, made the following report: Specimen received consists of the uterus, both tubes and ovaries and the appendix. The uterus is slightly enlarged with marked arteriosclerotic changes throughout its walls. The endometrium is thin, and rather leathery in consistency. Cervix is moderately lacerated and a small polyp springs from within the canal; benign. The right ovary is small, hard and wrinkled, atrophic in type. The tube is sealed at the fimbria, but is otherwise normal. Appendix removed prophylactically.

The left ovary forms a large cystic tumor which measures 18 × 14 × 11 cm., weighing 3¾ lbs. It is smooth in outline and unruptured. The fallopian tube is attached to the tumor. The cyst wall is thin and dark bluish gray in color. When the specimen was opened, 1400 c.c. of dark brown fluid were removed. Smears from the fluid contained cholesterin crystals and many blood cells. There were no dermoid elements such as hair, bone, teeth, or other epithelial structures found in the specimen. At the base of the tumor, near the pedicle, and growing firmly to the wall, but completely encapsulated, there were two masses of tissue that were in contact with each other. They were soft, pale red in color, giving the appearance of epithelial growths. The largest mass was 4 cm. in diameter and the small one 2.5 cm. They were well encapsulated and not a portion of the adjoining cyst. There were a large number of small, dark, bluish cystic formations beneath the capsule and in juxtaposition to the growth at this point.

Microscopic Sections: Microscopic sections of the encapsulated masses show them to be composed wholly of thyroid tissue. The specimen is characterized by large, fairly regular rounded spaces filled with amorphous, homogeneous substances (colloid material). The structure is composed of separate acini supported by connective tissue stroma. Most of the acini are lined by a single layer of granular epithelial cells with deeply stained nuclei. In other areas the acini are crowded close together and contain but little or no colloid. The dense connective tissue between the acini contains masses of undifferentiated cells which are probably young acini. In still other areas, between the glandular acini are cell masses without a central lumen or cavity, which are similar in appearance to fetal thyroid or fetal adenomatous tissue.

The evidence of moderate hyperplasia is probably due to the increase in number rather than increase in size of the acini.

Note: No studies were made to determine the amount of iodine in the thyroid tissue. A careful search was made for other teratomatous elements but none found.

The tumor was then sent to Dr. A. C. Broders at the Mayo Clinic. A letter from him states that the tissue in the ovarian tumor is definitely fetal adenoma of the thyroid type.

SUMMARY

While struma ovarii is considered a rare condition, it probably occurs more often than the literature indicates. The work of Pick, Plaut and others has been very helpful in determining the genesis of the condition. A study of the literature should inspire us to be on the alert for other cases and place them on record. As suggested by Morgen, it may be possible that the same endocrine disturbance that leads to a struma colli may also stimulate a goitrous development in the ovary.

The writer believes that the case herein reported is a true ovarian goiter, teratomatous in origin, and belongs to that group of cases where the predominant thyroid tissue outgrew the other elements.

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[For Discussion see p. 848.]

OTHER LABORATORY METHODS IN THE HANDLING OF THYROID DISEASE

G. D. MAHON, JR., M.D.

DALLAS, TEX.

IN taking up the question of diseases of the thyroid gland it is always well to spend a few minutes in a brief review of Plummer's classification. Prior to Plummer's classification of diseases of the thyroid the vast trouble came in considering, or in treating, adenoma of the thyroid with hyperthyroidism and exophthalmic goiter as one and the same thing. In 1911 Plummer suggested the following classification, which, for the most part, has been accepted for the last twenty years and more:

1. Colloid goiter
2. Exophthalmic goiter, or Graves' disease
3. Adenoma without hyperthyroidism
4. Adenoma with hyperthyroidism.

Colloid goiter is, a simple enlargement of the thyroid gland in which the acini of the gland fills with colloid. This disturbance, or physiological process, if it may be called that, begins around puberty and offers, as a rule, no trouble in diagnosis. Being largely a self-limited condition treatment ordinarily is not indicated. The only difficulty comes when this enlargement of the thyroid gland is found in an individual who shows certain signs that may be confused at the time with an early exophthalmic goiter, such as nervousness, tachycardia, and loss of weight. For the most part, however, this possibility can be ruled in or out with a basal metabolic reading.

Exophthalmic goiter, or Graves' disease, is a disease in which the thyroid gland unquestionably plays an important part, because in the presence of Graves' disease there is always present hypertrophy and hyperplasia of the thyroid gland. The symptoms consist of, loss of weight, loss of strength,

muscular weakness, increased susceptibility to heat, and finally, exophthalmos, which is present in from 85 to 90 per cent of cases.

Adenoma of the thyroid in which no toxic symptoms are present is simply an enlargement of the thyroid gland, producing no symptoms or ill effects other than its presence.

Adenoma of the thyroid with hyperthyroidism, commonly spoken of as thyrotoxicosis, is the fourth group. In this condition the same chain of symptoms is present as is present in the exophthalmic goiter, with the following variations:

1. The symptoms appear in the exophthalmic goiter within a very short time after, or simultaneously with, the hypertrophy of the thyroid gland, whereas in the adenoma the patient may have been the host of the tumor for a number of years. In the cases that Plummer saw the tumor had been present on the average of fifteen years before there was any indication of toxic symptoms.

2. Another difference is that, regardless of how severe the toxic symptoms may be in the adenomatous goiter, exophthalmos is not present except in the very small number of cases in which there is a possibility of the two conditions exophthalmic goiter and adenomatous goiter coexisting.

These two differences, existing in the two conditions, caused Plummer to speculate why the exophthalmic goiter produces exophthalmos and why the adenoma produces every other symptom except exophthalmos. He reasons that the adenoma produces toxic symptoms by an over-production of normal thyroxin, this over-production either taking place in the adenomatous tissue itself or in adjacent so-called normal gland tissue. At the same time he feels that the exophthalmic goiter produces its toxic symptoms by an over-production of normal thyroxin plus an additional production of an incomplete, or a more toxic, form of thyroxin than is present in the adenomatous group. Just what this substance is has not yet been determined, it being purely theoretical up to the present time. The pathologist is able to find in the exophthalmic goiter always some

degree of hypertrophy and hyperplasia present. On the other hand, the pathologist is never able to say, in the examination of an adenomatous goiter, whether that patient has toxic symptoms or not.

METABOLISM AND THYROID DISEASE

Up to the present time we have been more or less content in arriving at the status of thyroid disease by the taking of metabolic rates. Metabolism may be determined by direct or indirect methods. The direct method is not practical for clinical use, it being hard to elaborate, but having been checked frequently with the so-called indirect calorimetry the indirect method has been thought to suffice for all practical purposes. The indirect method consists of the determination of how much oxygen will be consumed by a given patient in a given time, taken under certain ideal conditions. To have this examination made a little more correct a determination of the carbon dioxide output may also be taken, which is done in many of the better laboratories. But for the most part even this refinement does not add much to the knowledge obtained of a given patient.

Granted that the above is all true, it seems that diseases of the thyroid have been pretty well settled, and certainly, for the most part, they have, yet there are certain conditions or possibilities that have not been settled, and it is of these that I wish briefly to speak.

1. Each year I see several patients who present themselves with an exophthalmic goiter in which a diagnosis with our present methods could not be arrived at until considerable time had elapsed. Is it possible to obtain other laboratory data that will materially aid in an earlier diagnosis?

2. We see several cases every year of adenoma of the thyroid in which the patient is not well, and in which there may be a heart complication i.e., there may be anginal or pseudo-anginal disturbances. There may be a sense of failing

health, with no laboratory methods up to the present time by which one can say with assurance that the thyroid is at fault. During the year we have operated on three adenomas of the thyroid, all with mild hypertension, two of them presenting cardiac disturbances, and yet repeated basal rates were entirely normal. Removal of the adenomas, however, has restored these individuals to what we think of as normal health.

3. In another group we see evidences of a definite exophthalmic goiter, with all the signs and symptoms ordinarily indicating surgical relief. Surgery is done. The usual amount of gland is removed, and then the patient will have a recurrence almost before he gets out of the hospital. This year I have operated on one patient three times, removing a small portion of thyroid at each operation. Can we develop other methods of study that will offer a more perfect prognostic aid? In other words, is there some pathological condition to be found in the gland to give us this information ahead of time? Could we, by examining the blood of this patient, have an inkling that he belongs to the recurring group? In trying to answer this question I have searched the literature for other laboratory methods that have been employed in the study of this interesting group of patients.

CHOLESTEROL IN HYPOTHYROIDISM AND HYPERTHYROIDISM

Luden, in 1918, found that by giving desiccated thyroid to a myxedematous patient the cholesterol value in the blood was found to decrease and the basal metabolic rate increase. More recently a rather extensive study has been made by Hurxthal, of Boston, and his conclusions are about as follows:

1. Postoperative myxedema is accompanied by hypercholesteremia.

2. Subtotal thyroidectomy may be followed by hypercholesteremia, without clinical myxedema. This is interpreted as a transient thyroid deficiency.

3. Subtotal thyroidectomy may be followed by low metabolic rates, without hypercholesteremia. In these cases myxedema is seldom found.

4. Roentgen therapy can produce transient hypercholesteremia with or without clinical myxedema.

5. Removal of part of a thyroid gland affected by chronic thyroiditis may be followed by hypercholesteremia and myxedema.

6. Thyroid deficiency produces myxedema and hypercholesteremia, but at times myxedema may be clinically imperceptible.

7. Hypercholesteremia, when not explainable on any other basis, may be considered as possibly of thyroid origin, and is a rational indication for thyroid administration.

8. The finding of hypercholesteremia, in the absence of its few other common causes, points more specifically to thyroid deficiency than does the finding of a low metabolic rate. Finding both renders the possibility of thyroid deficiency extremely likely.

9. The relationship between the blood cholesterol and the basal metabolism is usually reciprocal when they undergo change as the result of variations in the activity of the thyroid gland or thyroid substance in the body.

10. The blood cholesterol provides another variable which may be used as a guide in the treatment of thyroid disease.

This year we have made cholesterol determination in the following cases:

1. Twelve cases of severe hyperthyroidism.

2. Thirteen cases of moderately severe hyperthyroidism.

3. One case of moderately severe hyperthyroidism associated with a pregnancy.

4. Three cases of myxedema.

5. Three cases of postoperative myxedema.

We find hypercholesteremia in both spontaneous and acquired myxedema, at the same time finding no noticeable

change in cholesterol in the mild, moderately severe, and severe cases of hyperthyroidism.

STUDY OF THE BLOOD IN THYROID DISEASES

As early as 1918 Plummer did extensive blood studies, such as hemoglobin determination, blood count, white blood count, and complete differentials. Plummer's studies brought out only one blood change that stood out, and that was mononucleosis. Plummer states, however, that this being before they had careful metabolic studies, it was not determined whether the toxicity of the gland was a factor in the production of mononucleosis or whether it was present in both the mild and the severe type of hyperthyroidism.

Recently, we have had blood studies made on patients with marked hyperthyroidism previous to operation, at the highest level of their postoperative reaction, and again in three weeks' time. We are following the same sort of test as did Plummer, plus more attention being paid to the Schilling reaction, and Hill, of Baylor, is studying the individual cell with the idea of seeing if there are changes in the cell due to hyperthyroidism. Hill and one of his assistants, Miss White, have studied 12 cases preoperatively and postoperatively (forty-five hours). He was unable to draw any conclusions from this small group of cases.

DETERMINATION OF SEDIMENTATION SPEED OF ERYTHROCYTES AS METHOD FOR DIAGNOSIS AND FOR MEASURING THERAPEUTIC RESULTS IN DISORDERS OF THYROID

"According to Taterka and Goldmann the determination of the basal metabolism was formerly the method most commonly employed for determining the toxicity of disorders of the thyroid. However, this method is complicated and not always reliable. On the basis of the fact that in exophthalmic goiter there generally exists an increased sedimentation speed of the erythrocytes, the authors investigated whether there is a parallelism between the increased sedimentation speed and

the toxicity of the hyperthyroidism. They come to the conclusion that the determination of the sedimentation speed of the erythrocytes is a simpler and a more reliable method than the measuring of the basal metabolic rate. The sedimentation speed indicates the degree of toxicity, and it is particularly helpful to control the therapeutic effects of a treatment, especially of roentgen irradiation. On the other hand, this method may be used to determine the quantity of thyroid extract that is necessary for treatment of hypofunctioning of the thyroid, as in patients with myxedema or with adiposity."

Dr. Whitten, one of my associates, studied a few cases of hypothyroidism in 1932 and felt his findings were too variable to be of value.

BLOOD IODINE IN THYROID DISEASE

Coindet used iodine as early as 1820 in the treatment of goiter. Since that time iodine and the goiter problem have not been far separated. In 1900 Gley and Bourcet discovered iodine in the human blood. The quantitative determination of iodine in the human blood has been a very difficult technical problem, and it has been only in more recent times that any deductions could be made. The more accurate method for the determination of blood iodine was worked out by von Fellenberg and modified by Lunde. Last year Curtis and Davis, of Columbus, Ohio, did a rather exhaustive study of blood iodine and diseases of the thyroid gland, and made the following deductions:

> The iodine content of human blood is thus a measure of thyroid function. Demonstrable changes in the level of the blood iodine accompany those diseases in which thyroid function is altered. The fluctuation of the blood iodine during the course of thyroid disease under management is significant. The blood iodine is increased in thyroid hyperfunction. It is lowered in thyroid hypofunction. It is always elevated by any form of iodine medication. It would seem that the level of the blood iodine is controlled by thyroid activity. There are corresponding changes in the urinary excretion of iodine. This increases in thyroid hyperfunction. It likewise is increased on any form of iodine medication.

Simultaneous investigation of the basal metabolic rate, the level of the blood iodine and the urinary excretion of iodine reveals significant evidence of thyroid activity. The significance of the blood iodine in thyroid disease is similar to that of the blood sugar in diabetes mellitus and to that of the blood calcium in parathyroid disease.

We have made an attempt to do blood iodide determinations for the last several months, but do not feel that at the present time our technique has been perfected enough to warrant any deductions being made. I expect to report the present work at a later time.

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DISCUSSION ON PAPERS OF DR. SANDERS AND DR. MAHON

DR. FRANK H. LAHEY, Boston Mass.: I believe we have something in blood iodine determinations which offers possibilities in the diagnosis of hyperthyroidism other than basal metabolism tests. The work has as yet not been extensive enough to make any positive statements, but I am glad of the opportunity to present it to you so that Dr. Mahon may take it back and use it in his clinic and tell us whether it works out satisfactorily with him.

First as regards blood cholesterol, Dr. Hurxthal is the man who in our Clinic did the work on blood cholesterol. He has done blood cholesterols on about 1800 of these patients and has published several excellent papers on this subject. It has proved of little practical value in hyperthyroidism but of great value in hypothyroidism. It is, we believe, an even better criterion of thyroid activity in hypothyroidism than is basal metabolism.

I wish to present to you some of our experiences with the blood iodine determinations.

I present first some figures as relates to blood iodine determinations in hyperthyroidism to lay the ground work for illustrating the iodine tolerance test. In 22 cases of toxic goiter there were only 3 with blood iodines below the normal level and all others ran up into the ranges of high micrograms of blood iodine per 100 c.c. The average blood iodine in a group of patients with hyperthyroidism was 37 micrograms of iodine per 100 c.c. or three times the normal micrograms per 100 c.c.

The iodine tolerance test was devised and is employed by Mr. H. J. Perkin in our Clinic. It is possible to establish what is the normal base line for the blood iodine following the administration of iodine approximately 10 to 12 micrograms of iodine. We give 10 minims of Lugol's which represent 72 milligrams of iodine. We put the patient on a breakfast of coffee and toast, keep him in bed the night before and then collect the blood at intervals up to seven and a half hours afterward. You must remember that the patient with hyperthyroidism has a thyroid gland which is thirsty for iodine. It will take up the administered iodine avidly and you will not then have the iodine in the blood which you will have in patients with a non-hyperplastic gland.

In the patient who has hyperthyroidism and a hyperplastic gland the iodine curve in an iodine tolerance test is constantly below the normal base line 12 micrograms of iodine. That is due to the fact that the gland is so thirsty for iodine. You do not find it in the blood because it is accepted and stored by the gland. In the patient who does not have hyperthyroidism and so no gland affinity for iodine after the administration of iodine, the blood iodine promptly passes above the normal base line because there is no thirst in the normal thyroid for iodine. With an exophthalmic goiter we have the acceptance of iodine by the hyperplastic gland and the typical low blood iodine curve with it. With the normal gland, we have the rejection of iodine with the typical high blood iodine curve with it.

I think that here is a possible delicate test to demonstrate hyperplasia in the gland, and a possible differentiating point in the borderline cases. It is true that blood iodine tests have been considered inaccurate but Mr. H. J. Perkin, the biochemist in charge of this investigation in the Clinic, has devised and published a microdetermination method for blood iodine which is accurate, and I believe this test will prove worth while clinically.

Dr. Wm. T. Black, Memphis, Tenn.: I wish to refer to Dr. Sander's paper and to say a few words regarding the etiology of ovarian tumors. I think ovarian tumors are impossible in many instances to explain. We know that in many instances they develop from the germinal epithelial cells. We also know that the etiology of arrhenoblastoma can be explained, as we know that in early fetal life the gonads are asexual and you cannot tell whether it is an ovary or a testicle. If a testicle develops, the ovarian

structures regress or diappear entirely, but in some instances male remnants are left and if a tumor develops we have the characteristics of the male.

In the ovarian thyroid tumor, which has been splendidly described by Dr. Sanders, the etiology is probably teratomatous in character. The etiology can probably be better explained in this manner, as teratomata are derived from sex cells. In studying the etiology of teratomata it was formerly thought that they might develop pathogenetically, as some of the lower forms of life have been made to develop. Lower forms of life have been developed experimentally in this manner. We know that we have in the beginning of the development of the ovaries the germinal epithelium growing inwardly and forming the follicles, in which ova develop. Only a few of these follicles ever grow to maturity (the Graffian follicles). Considering then that there are various stages in the developmental changes in the ovum and that follicles often reach a certain age then begin to become regressive in nature; therefore, it is possible that we have immature ovum occurring at various ages and the stimulation of some toxin or inflammatory condition may stimulate this remnant of an ovum to develop into any sort of a teratomatous tumor formation.

Just why we have the predominance of thyroid tissue probably cannot be satisfactorily explained, but neither can we explain why we have developed bone and other structures in some and very embryonic structures in others. It may be possible that the same hormone or toxin which causes the development of thyroid gland tissue or causes a tumor formation, may act in the same manner upon thyroid tissue which was present in the beginning of an ovary and develop a thyroid tumor of the ovary.

DR. G. D. MAHON (*Closing*): I wish to thank Dr. Lahey for elucidating this subject much more clearly than I was able to do. The iodine curve, if it could be perfected, and it looks as if it could, may mean to us in the study of thyroid conditions what the glucose tolerance curve means in the diabetic patient.

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